

CAUSE AND CONTROL OF AUTOMOTIVE TRAUMA*

DAVID C. VIANO, Ph.D.

Principal Research Scientist
Biomedical Sciences Department
General Motors Research Laboratories
Warren, Michigan

TRAUMA is a public health problem with consequences equal to those of such major diseases as cancer, cardiovascular disease, and stroke, and is similar to disease in many respects.¹ Trauma alters the body's normal function and structure in characteristic and often predictable ways, but its causes are known to only varying degrees. Stedman² defines disease as an "entity characterized usually by at least two of these criteria: a recognized etiologic agent (or agents), an identifiable group of signs and symptoms, or consistent anatomical alterations."

Using Stedman's terms, the "agent" of trauma may be a sharp object or missile causing penetration of body tissues, a blunt object causing direct compression and rupture of tissues at the impact location, or abrupt changes in motion causing tissues to stretch and to separate under their own weight. An example of the latter is the sudden stopping of the head, where injury to the brain may occur as its motion lags behind that of the skull, causing brain tissues to shear and stretch until vessels are torn or neurons damaged. In all three situations the severity of the injury is related to the energy associated with the penetration, blunt impact, or acceleration environment. Trauma also has identifiable "signs and symptoms," including hemorrhage from rupture or laceration, loss of consciousness from brain damage, loss of motor function from spinal cord injury, and shock. Finally, trauma can involve a wide range of "anatomical alterations," and typical patterns of injury have been identified and studied in both clinical and experimental settings.

As with a disease, an understanding of the etiology of trauma is an important step toward its prevention and control. Yet much is still to be learned about trauma mechanisms, particularly with regard to severe internal organ

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and tissue damage from nonpenetrating impact. Areas of current research include the biomechanics and pathophysiology of anatomical lesions, both at the gross and the microscopic level, and associated functional changes within the body. Knowledge about these lesions is critical, not only for their own prevention and treatment, but because they initiate the serious effects of trauma, especially the debilitating changes to central nervous system tissues.

Although trauma is generally like a disease in definition, there are interesting differences in the opportunities for medical and engineering intervention (Figure 1). Cancer, for example, affords little chance to affect its lesions immediately before or after diagnosis. In contrast, the effects of trauma's potential agents can be significantly mitigated if occupant protection devices are in place during impact and if emergency medical care is available in the minutes following. Cardiovascular disease and stroke can also benefit from treatment during the acute phase, but, unlike trauma, prior intervention through electronic monitoring or medication is only practical for a few targeted patients. Trauma and these major diseases all share similar opportunities to minimize their incidence through preventive programs that may alter the environment and habits of the potential victims as well as opportunities to reduce long-term disabling effects through treatment and rehabilitation.

In the case of trauma, and particularly that occurring in the automotive environment, the preventive and protective approaches have an enormous potential for effective intervention. Some of these approaches, such as elimination of drunk driving, are consistent with good overall health practices. Others, such as the use of seatbelts, are already accepted norms among large segments of the population. The effectiveness of protective measures available in the potential impact environment itself, however, depends on an understanding of trauma mechanisms, the development of technology to take advantage of this understanding, and a public willing to take advantage of this technology.

This paper summarizes what is known about why automotive crashes occur, how and why injuries occur in these crashes, how devices and structures in the vehicle work to prevent or to reduce these injuries, and the limitations and trade-offs inherent in the overall effectiveness of these protective systems. A case is then made for a more balanced approach to trauma prevention and control, involving cooperation and consensus among a variety of organizations and agencies as an effective way to approach this significant public health problem in the future.

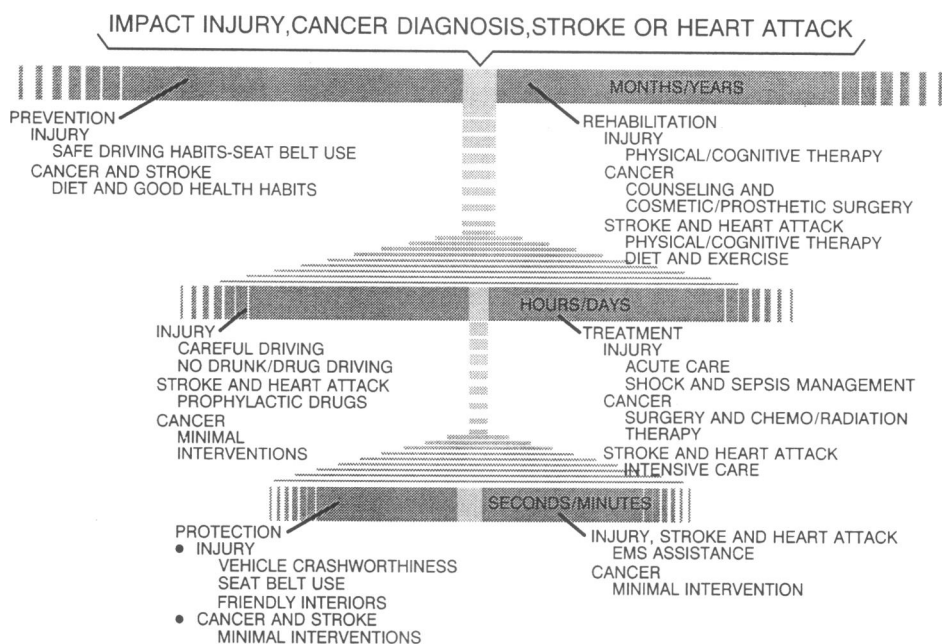


Fig. 1. Four principles of injury control: prevention, protection, treatment, and rehabilitation at the time points in which they are most effective. Examples of intervention are given for impact injury, cancer diagnosis, and stroke or heart attack.

Causes of Automotive Crashes

If crashes could be avoided altogether, there would be little need for elaborate occupant protection structures or treatment and rehabilitation strategies. While such a condition is very unlikely, a reduction in crashes is attainable if it is first known why they occur. For many years, the common assumption was that mechanical defects in vehicles caused most collisions and loss-of-control events. Research into the causes of actual crashes, however, has proved this far from accurate.

Treat³ conducted an in-depth investigation of factors causing automobile crashes. The investigation analysed more than 2,000 traffic accidents whose types and severities represented the total population of police-reported crashes. About three quarters of them involved only property damage, about one quarter involved injuries to occupants, and only about 1% involved deaths. On-site investigation of the crashes allowed an assessment whether specific human, environmental, or vehicular factors were definitely, probably, or possibly involved as either causal or severity-increasing factors.

A factor was considered causal if, in its absence, the accident would not have occurred. This definition, however, allows several factors to be identified as causing a single accident, as was usually the case in those investigated. A severity-increasing factor was one that was not necessary for occurrence of the accident, but its removal would have lessened the severity of the initial impact in terms of speed. Assessment of factors associated with each crash involved two major steps: identify relevant deficiencies of drivers, vehicles, and driving environment present in the accident sequence and assess whether the accident would not have occurred had each deficiency been corrected to its minimally acceptable state.

The results of the investigation indicated that human factors were the most frequently implicated of the three categories, and vehicle factors were the least frequently associated with motor vehicle crashes (Figure 2). Human factors were implicated as a definite causal factor in 70.7% of the accidents, environmental factors in 12.4%, and vehicle factors in 4.5%. These same three categories were implicated as definite, probable, or severity-increasing factors in a higher fraction of the crashes. Although more than one general category as well as a number of factors within each category were found for most of the accidents, in 26% of the cases investigated no definite cause was established. In these cases, however, at least one probable cause was always identified.

In 57.1% of the cases human factors were the sole cause of the accident. In another 26.4% a combination of human and environmental factors was determined as responsible. In 6.2% of the crashes there were both human and vehicular causal factors, and in 3.3% environmental factors only were causally associated with the crash. Other factor combinations accounted for even lower fractions of accident causation.

Two general categories of human factors were identified as important to an automobile crash. Human errors were responsible in the highest fraction of crashes, with recognition and decision errors, improper look-out, inattention, and the consequences of false assumptions and excessive speed being the most frequently associated with the accident. In a lower fraction of the crashes, human conditions were found to be causal factors in accidents. These conditions fall into two general categories: one related to impairment from alcohol, fatigue, drugs, or emotional problems and the other related to unfamiliarity with the roadway or driving inexperience.

Environmental factors responsible for crashes were defined as roadway design and condition, visibility, and all other precrash factors not related to the driver or vehicle. Environmental factors most frequently associated with

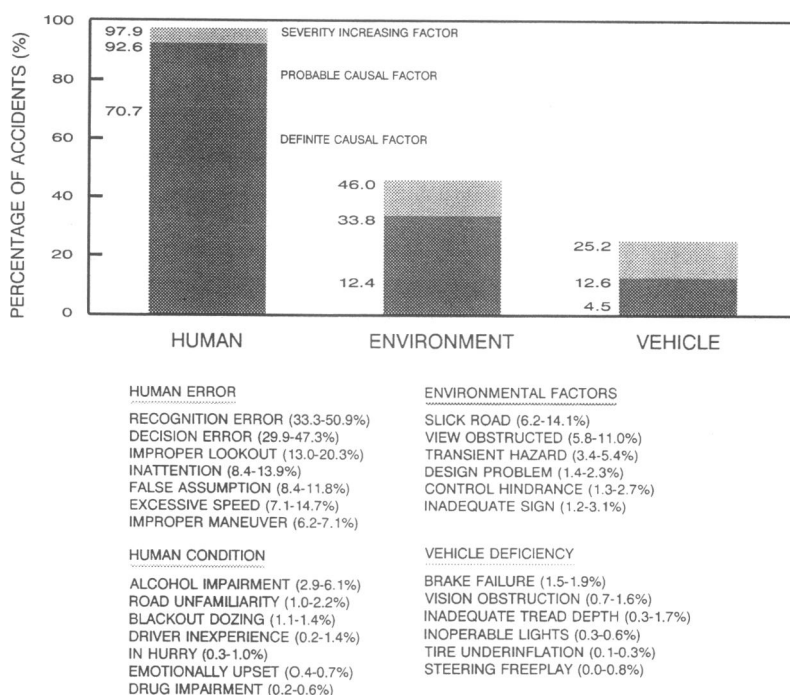


Fig. 2. Human, environmental, and vehicle factors causing motor vehicle crashes (data from Treat³)

crashes were slick roads, obstructed view of the roadway, and transient hazards.

Vehicle factors were overall the least frequent cause of crashes, and, among these vehicular deficiencies, most were related to poor maintenance. The largest vehicular factor associated with motor vehicle crashes was gross brake failure, causing a sudden and complete loss of brake function in older vehicles equipped with single-chamber master cylinders. These failures resulted primarily from brake-hose damage or loss of pressure at the wheel cylinders. Most tire and associated vehicle handling problems involved inadequate tread depth and underinflation. Vision obstructions from ice, frost, or water were also classed as vehicle-related factors.

In summing up causal factors associated with motor vehicle crashes, human error and impairment are clearly involved to a high degree, with less contribution from environmental factors and still less from vehicle factors. Among the latter, poor maintenance or aging of vehicle components are most likely to be involved. The obvious conclusion from this in-depth analysis is

that vehicle design is not a particularly significant factor in accident causation, and that the issues of accident avoidance or reduction fall primarily within the area of human behavior. Given the difficulties inherent in trying to change human behavior and the limits of warning systems and other technical aids in accident avoidance, it must be accepted that crashes will continue to occur. The focus for the vehicle, then, is the development of means to minimize the risks of injury associated with motor vehicle crashes. The following sections address this issue.

Injury Biomechanics

The design of automotive occupant protection systems requires understanding of both the mechanisms of injury and human impact tolerance. Injury mechanisms are the physical processes that result in tissue damage and/or functional impairment, while human impact tolerance refers to the levels of unusual stresses or loads that the human system can withstand with little or no injury. This field of research is called injury biomechanics⁴ and, as such, focuses on nonpenetrating types of injuries that can occur among both restrained and unrestrained occupants of highway vehicles involved in crashes. With sufficient information about injury mechanisms and tolerance, engineers can develop systems that will maximize occupant protection across the full range of crash configurations. This is achieved through the development of realistic anthropomorphic test devices (dummies) and criteria to evaluate their engineering measurements, which in turn are used to assess the effectiveness of protective systems in the development stage as well as in actual crashes.

TRAUMA MECHANISMS

Impact between the human body and an external object can cause compression, stretching, and other deformation of tissues beyond their recoverable limits. In an inorganic system this would be called mechanical failure; in the human system it is called trauma. In some cases, there may be no apparent physical damage, but functional changes may occur nonetheless.

Two basic mechanisms are associated with blunt, nonpenetrating impact injuries: localized loading and acceleration in the direction of loading. In the automotive environment the primary collision between the vehicle and whatever it strikes is followed by an impact between the occupant and the inside of the vehicle. This local loading of a part of the body against the instrument panel or even a seat belt is often referred to as the "second collision." There

is also, however, a “third collision” between soft tissue and skeletal structures that takes place inside the body itself as it is being stopped by the vehicle interior or restraint system. The contribution of these two types of impacts to the injury process differs depending on the body region and the severity of the impact, as described below. The basic function of an occupant protection system is to reduce the severity of these impacts and thus their potential for causing injury.

Impact to the face can directly cause laceration from both blunt and sharp surfaces, and severe blows can cause skull fracture. At the same time, however, the impacted head experiences abrupt deceleration during which the rigid skull slows down before the softer brain.⁵ This results in relative motion between the brain and skull and causes an internal impact on the side toward the external load, deformation within the brain itself, and tearing away from the skull on the opposite side. With a sufficiently severe impact, these processes result in vascular and neural damage from compression, stretching, and shearing of tissues. The greatest concern regarding closed head impact, then, is the injury potential from impact acceleration. In the abdominal region, injuries from blunt impact may result from either direct deformation of soft tissues or relative internal motion. Major laceration of the liver at the vascular junction is evidence of the latter mechanism. Injuries from local loading are more common, however, and can occur before significant whole-body deceleration.⁶

The primary mechanism of thoracic injury is direct compression of the rib cage in combination with deformation and stretching of internal organs and vessels during blunt impact.⁷ When chest compression exceeds the tolerance of the rib cage, ribs fracture and internal organs and vessels can be ruptured. In some impacts, however, internal injury can occur without skeletal damage. This can happen particularly during high-speed loading and is due to the viscous or rate-sensitive nature of human tissues and thus their different responses to low- versus high-speed impact. When the organs or vessels are loaded slowly, the input energy is absorbed gradually through deformation. When loaded rapidly, however, the viscous tissues cannot deform rapidly enough. Instead, they develop high internal pressures and can rupture before the ribs have deflected much at all. The ability of an organ or other system to absorb such energy rapidly without compressive failure is called its viscous tolerance. Internal organs and vessels can also be torn from their points of attachment during thoracic acceleration and an associated rapid motion of the undamaged rib cage, but this is a rare event.

For both penetrating and nonpenetrating impacts, the primary factors that

determine the type and severity of injury are the amount of body area over which the impact energy is spread and the speed of the impacting object. Effective restraint systems not only spread the impact energy over the strongest body structures but markedly reduce the contact velocity between the body and the surfaces stopping its motion. To assist in the design of such systems, efforts have been made to quantify levels of injury and to establish numerical relationships between measurable engineering parameters such as force, acceleration or deformation, and these injury levels. These relationships are called injury criteria, and the primary concepts are discussed below.

ACCELERATION TOLERANCE CRITERION

Stapp⁸ conducted a series of rocket sled experiments that demonstrated the effectiveness of belt-restraint systems in achieving high tolerance to long-duration, whole-body acceleration and thus improved the protection of military personnel exposed to rapid but sustained deceleration. Information from these experiments and from other tests enabled Eiband⁹ to demonstrate that whole-body deceleration tolerance increased as the duration of the exposure decreased. This led to a well-accepted deceleration tolerance curve (Figure 3) that for the first time linked tolerance information generated in regimes differing by orders of magnitude in duration of deceleration. This tolerance curve was based, however, on peak sled accelerations and total time duration rather than an average of peak accelerations measured on the test subjects themselves. Even with this limitation, the data did provide useful early guidelines for the development of crash restraint systems for both military and civilian personnel. The analysis also indicated that rate of onset affected acceleration tolerance, in that higher peaks could be tolerated if they were reached over a greater period of time.

The basis for the whole-body acceleration tolerance criterion is Newton's second law: that the acceleration of a rigid mass is in proportion to the force on the moving mass, or $F=ma$. Although the human body is not a rigid mass, as will be discussed later, a well-distributed restraint system such as the harness used in the rocket sled tests makes the thorax at least respond as though it were fairly rigid. Thus, the greater the acceleration, the greater the force on the body and the greater the risk of injury. Ability to withstand higher accelerations of shorter duration implies that tolerance is also related to the transfer of momentum because an equivalent change in velocity can be achieved by increasing the level of acceleration and decreasing its duration or vice versa as $\Delta V = a\Delta t$. The implication for occupant protection systems is that the risk of injury can be decreased if the crash deceleration can be

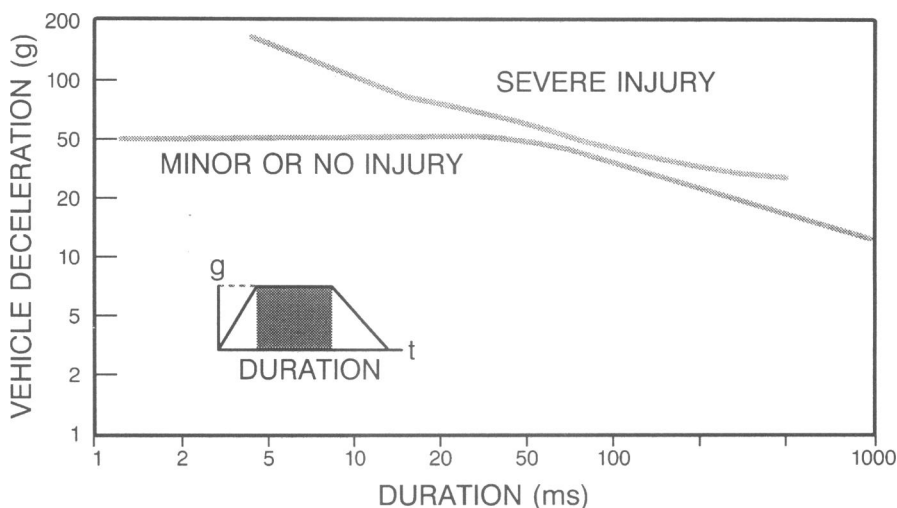


Fig. 3. Whole-body human tolerance to vehicle deceleration based on impact duration (re-drawn from Eiband⁹).

extended over a longer period of time. This can be achieved by increasing the body's stopping distance through the use of crushable structures and seat belts, to be discussed in a later section.

FORCE TOLERANCE CRITERION

In the early 1960s automotive safety engineers were seeking information on the tolerance of the thorax to applied force. This information was needed for the development of the energy-absorbing steering system. The energy-absorbing column included a device that limited the force that could be applied to it before the structure would deform and absorb energy as it compressed. Unfortunately, the available human tolerance data did not provide specific information on the appropriate force at which the system should begin to deform or to yield. The basic objective for the system was to design the yield force to be as high as practical while being consistent with human tolerance, to take maximum advantage of its energy-absorbing capacity and thus to extend its range of protective capability to higher severity crashes.

Faced with uncertain information on the force tolerance of the chest, a collaborative program was undertaken between General Motors Research Laboratories and Wayne State University to develop a crash-simulation facility and to conduct experiments on human tolerance in automotive crash situations. The first series of experiments by Patrick et al.¹⁰ involved sled tests with embalmed human cadavers to simulate the response of an unre-

strained occupant interacting with load-measuring surfaces. Head, chest, and knee contacts against padded load-cells provided the first information on human tolerance to impact force. The resulting data on force tolerance of the rib cage provided the necessary information to design the energy-absorbing element in the steering column biomechanically.

Subsequent experiments by Gadd and Patrick¹¹ with a prototype energy-absorbing steering system confirmed that a 3.29 kN (740 lb) maximum force on the sternum from the wheel hub and an 8.00 kN (1,800 lb) maximum distributed load on the shoulders and chest resulted in column compression with only minor risk of rib fracture to the cadavers. These load levels were thus considered conservative thresholds of injury in situations with well-centered impacts using a thoracic-force injury criterion.

COMPRESSION TOLERANCE CRITERION

In the course of the cadaver impact experiments it was observed that the concept of whole-body deceleration and force did not adequately describe the tolerance of the chest to blunt impact nor the risk of internal organ injury. This is because force acting on a deformable body generates two simultaneous responses: compression of the thoracic structure and acceleration of its masses. The neglected mechanism of injury was compression of the chest, which caused the sternum and ribs to bend and possibly fracture when their bending tolerance was exceeded. Force is therefore not a sufficient indicator of injury risk by impact because it cannot discriminate between the two potential responses and because the acceleration mechanism is less related to thoracic injury than is compression.

This was confirmed by Kroell,¹² who analyzed a large series of blunt thoracic impact experiments and found that the maximum acceleration of the thoracic spine was a poor indicator of injury potential for frontal thoracic loading, and that compression, in terms of maximum percent change in chest thickness, was a superior predictor of chest injury (Figure 4a). Kroell's tests with human volunteers showed that chest compression up to 20% during moderately long-duration loading produced no detectable injury, and was fully reversible. Impact tests with human cadavers at levels of compression greater than 20% showed that, as the compression increased, the risk of skeletal fractures in the rib cage increased. At 40% compression, multiple skeletal injuries occurred, indicative of flail chest. Further analysis of the data by Viano¹³ found that, as the protective capability of the rib cage was destroyed by multiple fractures (Figure 4b), direct loading of vital organs occurred, producing injury to the heart, lungs, and great vessels. Using the

Kroell data, Neathery et al.¹⁴ developed a criterion of maximum chest compression for evaluating the risk of injury from blunt frontal impact. This criterion indicated that an average-size middle-aged male could experience a chest compression of 8.8 cm (3.5 in) with moderate but recoverable injury.

Evaluation of the time-varying force and compression response of the chest in human cadaver experiments also provided information on the dynamic stiffness of the thorax. Experimental data on biomechanical response also allowed the development of corridors predicting the impact response of the 50th percentile male and other sized cadavers. These data were pivotal to the development of an anthropomorphic test device with improved thoracic biofidelity,¹⁵ which ultimately led to the Hybrid III dummy chest used today. Its improved thoracic structure and response characteristics have allowed the Hybrid III to be identified as the most appropriate available test tool for the evaluation of vehicle components associated with blunt chest impact, such as the energy-absorbing steering system, over a wide range of sled and crash test environments.¹⁶

VISCOUS TOLERANCE CRITERION

Over the years, research on the mechanisms of soft-tissue injury has made it increasingly evident that the body cannot be considered a rigid structure, and thus an injury criterion based on whole-body acceleration is an inadequate predictor of injury risk. The body is, instead, a deformable structure, and, under conditions of low impact speeds (<5 m/s or 11 mph), rib-cage tolerance and risk of crush injury can be evaluated using a compression criterion. This criterion is particularly applicable to belt-restrained occupants in frontal crashes, in which the relative velocity between the occupant and a reasonably snug restraint system is small, as is, therefore, the rate of chest compression. For high thoracic compression rates (>5 m/s), however, which are typically experienced by unrestrained occupants or those in high-speed side impacts, the maximum compression criterion does not adequately address the viscous properties of the chest and thus risks of soft-tissue injury. For high-speed thoracic impacts, both the percentage compression and the velocity of deformation are important parameters relating to injury.

Insight into the mechanism of soft tissue injury in high-speed impact can be obtained from research on blast-wave injury. Jonsson and Clemmedson¹⁷ conducted a comprehensive series of lateral thoracic impact experiments on rabbits that confirmed the observation that chest compression tolerance to lung injury is not a fixed maximum value for a wide range of impact velocities

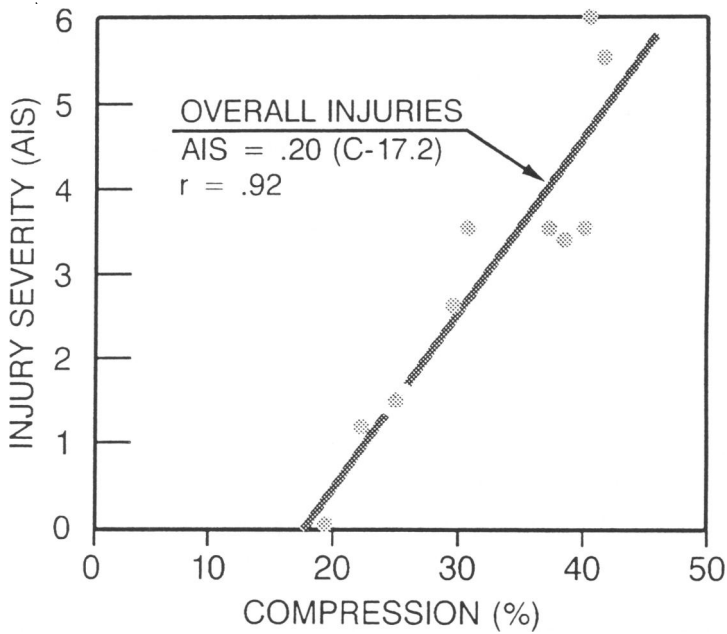
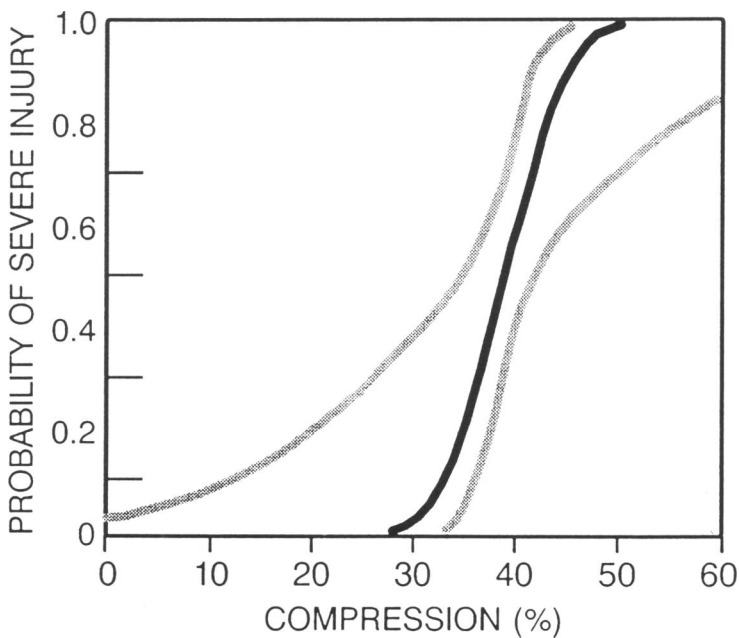


Fig. 4a. Injury severity from blunt impacts of human cadavers as a function of the maximum chest compression (redrawn for group averages of similar tests from¹³ and risk of severe injury as a function of maximum compression based on Logist analysis of the data in ¹³; Chi-square=23.88, $p=0.000$, $R=0.629$.



but rather varies inversely with the velocity of thoracic impact.

The concept was further studied relative to the abdomen by Lau and Viano¹⁸ using a velocity range of 5 to 20 m/s (11 to 45 mph), which are potential body impact speeds in high-speed automotive collisions or sports injury environments. The liver was the target organ, and the test was set up to attain a maximum level of 16% abdominal compression, well within the range of tolerable compression for a human volunteer in low-speed (0.4 m/s or 0.9 mph) loading. Using a varying rate of abdominal compression with the rabbit model, the experiments verified an increasing severity of liver injury as the velocity of abdominal compression increased (Figure 5a). Thus, rate of compression is an important factor in soft tissue injury. Subsequent blunt thoracic impact experiments on other animal models and organ systems substantiated the interrelationship between magnitude and velocity of chest compression. These two factors were found critical to the severity of both skeletal and internal thoracic injury including the likelihood of ventricular fibrillation.¹⁹ Similar findings regarding the importance of compression velocity were identified by Stein et al.²⁰ in a series of open thoracic impacts of the canine heart, in which the risk of fatal arrhythmias increased with the velocity of cardiac compression.

The previous observations led Viano and Lau²¹ to propose the concept of a viscous tolerance criterion for soft tissues in the body, which addressed the combined influence of compression percentage and velocity of deformation. The relationship between deformation velocity (V) and compression (C), defined as $VC = [V(t)*C(t)]$, is called the viscous response and is in fact a measure of the energy dissipated by rate-dependent (viscous) elements in the thorax.²² A large series of frontal thoracic impact experiments on rabbits was conducted with velocities of compression in the range of 5 to 22 m/s (11 to 49 mph) and maximum thoracic compressions of 4% to 55%. Using a threshold of critical/fatal injury, the experiments confirmed a velocity and compression sensitivity in tolerance to chest impact. Although the chest could withstand 50% compression for a 5 m/s rate of deformation, the tolerance decreased significantly to 20% at velocities of 20 m/s (Figure 5b). Further analysis of the data showed that the maximum of the product of V and C was the best predictor of critical injury risk. This product (VC_{max}) is the maximum viscous response of a system.

The series of blunt chest impacts on unembalmed cadavers conducted at the University of California at San Diego¹² was reanalyzed by Viano and Lau²² to determine the viscous response of the chest. Compression was determined by analysis of high-speed photographs of the impact event, and

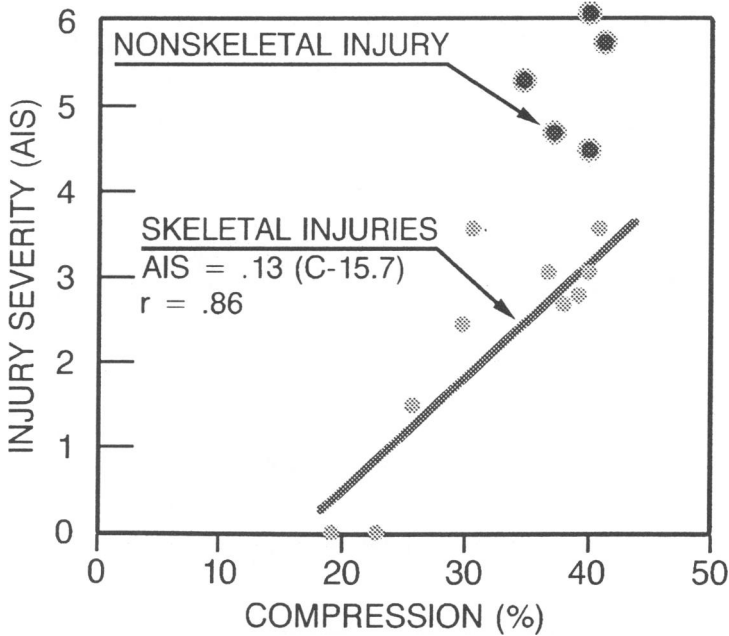
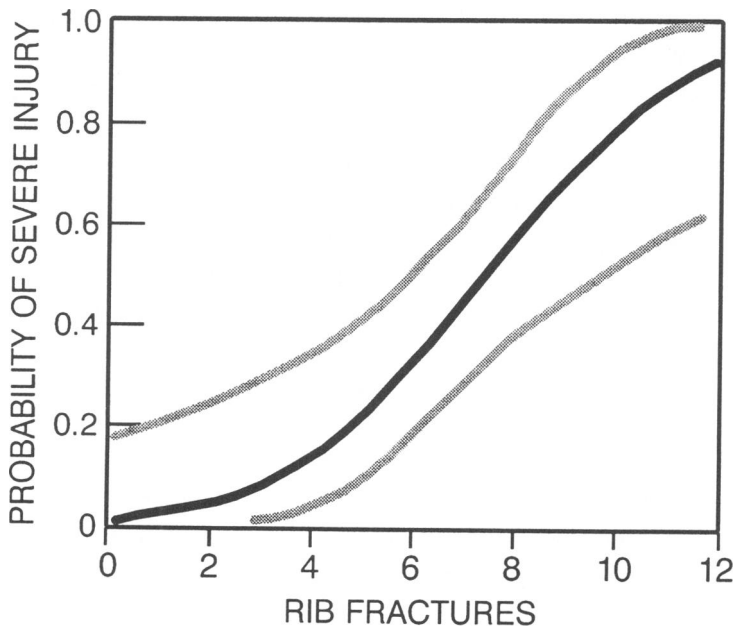


Fig. 4b. Severity of skeletal injuries and incidence of internal organ injury as a function of maximum chest compression for blunt impacts of human cadavers (redrawn for group averages of similar tests from¹³) and risk of severe injury as a function of the number of rib fractures based on Logist analysis of the data in¹³: Chi-square=17.69, p=0.000, R=0.526.



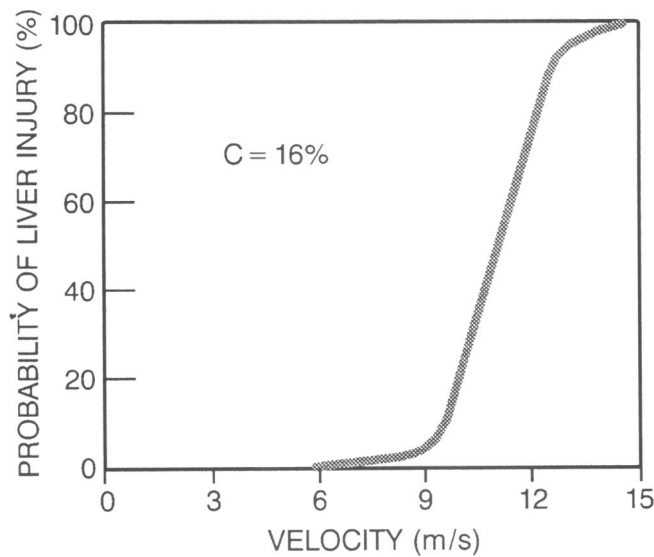


Fig. 5a. Risk of severe liver laceration as a function of the velocity of blunt abdominal deformation reaching a 16% maximum compression (Logist analysis of data in¹⁸: Chi-square=23.61, $p=0.000$, $R=0.774$).

the time histories of the responses were used to determine the viscous response of the cadavers. The peak viscous response was found to occur 20 ms earlier than maximum chest compression in the higher speed impacts of the cadavers. Kroell¹² has shown that rib fractures occur progressively with compression, the first fracture occurring as early as 9.2 ms and the second at 13.6 ms. In an event such as this, which takes 30 ms to reach maximum compression, peak viscous response occurs at about the same time as the initiation of skeletal damage. The maximum viscous response was correlated with the risk of critical injury using probit analysis (Figure 6a), and a good correlation was found between the maximum viscous response and the risk of critical/fatal soft tissue injury.

In more recent experiments on upper-abdominal injury from steering wheel contact, the maximum viscous response proved the best predictor of liver injury severity in the pig as well as time of injury occurrence.^{6,23} It also described more clearly the important biomechanical responses of the abdomen during steering wheel contact. These studies showed that liver injury by steering wheel contact occurred at the same time as the peak of the abdominal viscous response, which was prior to maximum compression of the abdomen

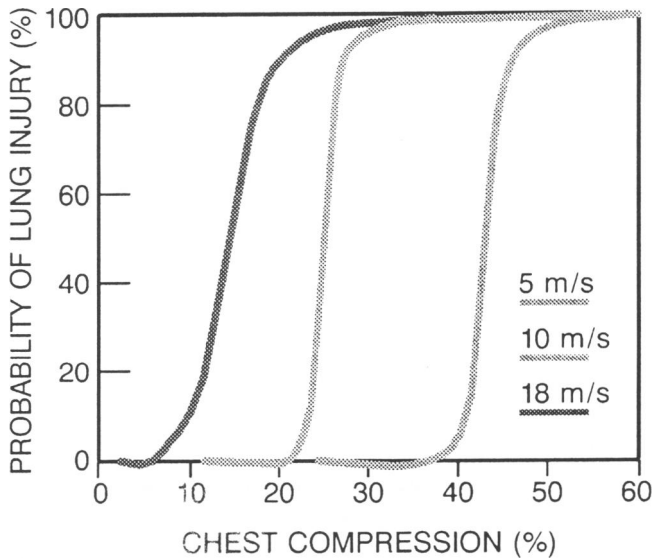


Fig. 5b. Risk of serious lung injury as a function of the maximum compression of the chest at three velocities of deformation (Logist analysis of data in²¹: $V=5$ m/s: Chi-square=15.45, $p=0.000$, $R=0.779$; $V=10$ m/s: Chi-square=17.32, $p=0.000$, $R=0.798$; $V=18$ m/s: Chi-square=21.71, $p=0.000$, $R=0.791$).

or maximum acceleration of the spine. In the pig, $VC_{\max} = 1.4$ m/s is the effective dose for 50% critical liver injury (Figure 6b).²⁴

A major goal of biomechanics research is to quantify the response and tolerance of the human during impact in order to develop an understanding of how the body can best be protected using established principles of load distribution and energy absorption. This information is transferred to the laboratory, where simulations of crashes and humans, in the form of anthropomorphic test devices, are used to develop, assess, and refine countermeasures that reduce the risk of impact injury, much as drugs are developed and tested to control arrhythmia or high blood pressure.

The following section describes the progress and advances made in occupant protection systems in recent years as well as the challenges in designing such systems for a full range of crash speeds and configurations. These difficulties would be diminished, however, with a more complete understanding of the injury process, which can best be obtained through a coordinated and balanced program of engineering and medical research on impact injury biomechanics.

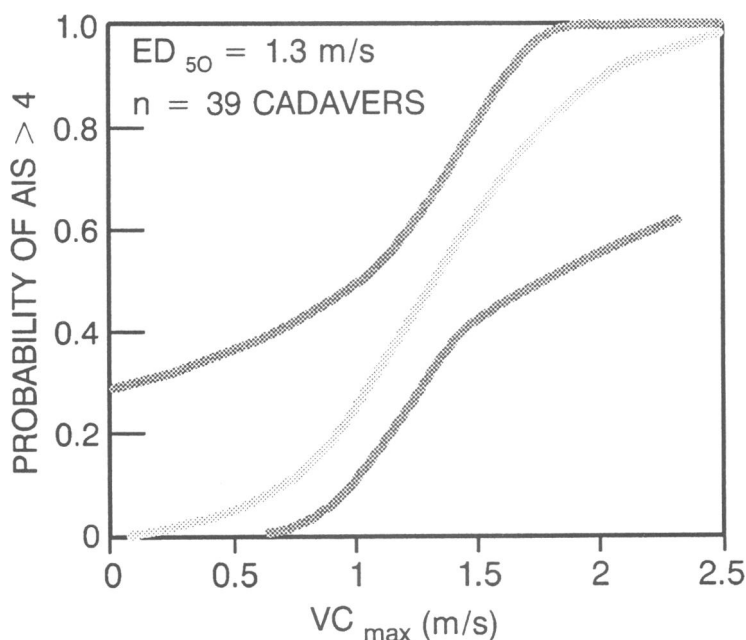


Fig. 6a. Risk of severe chest injury from blunt impact of human cadavers as a function of the maximum viscous response (Logist analysis of reanalyzed data from^{12,22}; Chi-square=16.85, $p=0.000$, $R=0.518$).

Occupant Protection Technology

The theory and application of vehicle occupant protection involves three aspects: vehicle crashworthiness, friendly interiors, and restraint systems. Each can contribute separately, but the most benefit can be achieved if all are combined in a safety system package. This section describes how each functions to reduce injury risk as well as how they interact in combination. Finally, the complexities of designing occupant protection systems for both high- and low-speed impacts are discussed along with the upper limits on the protective capabilities of these systems.

VEHICLE CRASHWORTHINESS

The earliest years of automotive safety technology, from about 1930 to 1950, emphasized structural integrity of the passenger compartment to contain the occupant in frontal and rollover crashes. Figure 7a shows where we were in 1929. In the 1960s the concept of energy management through crushable front-end structures was added. This combined approach attempted to

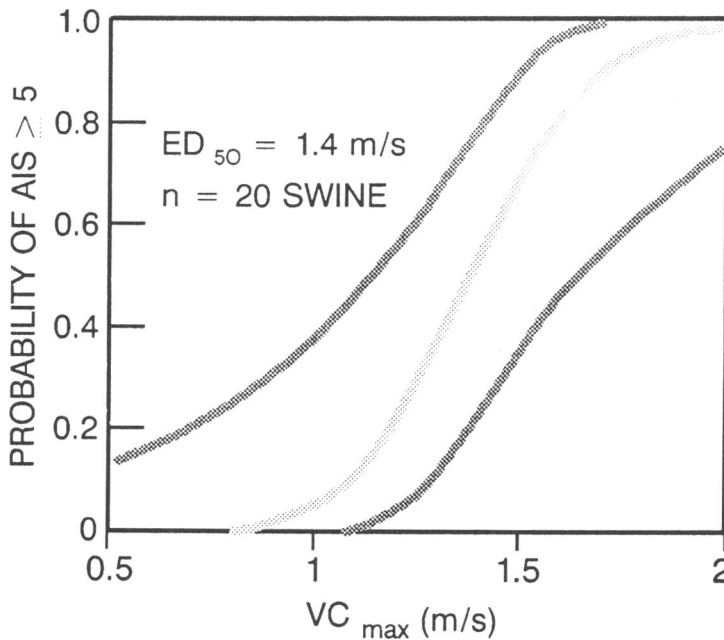


Fig. 6b. Risk of severe abdominal injury from blunt impact of anesthetized pigs as a function of the maximum viscous response (Logist analysis of data in⁶ and²³; Chi-square=27.52, $p=0.000$, $R=0.963$).

preserve the occupant's space, or "room to live," while the vehicle's crushing structures absorbed crash energy, lengthened the stopping time and distance of the passenger compartment, and thus reduced impact accelerations acting on the occupant. Achieving such crashworthiness in a vehicle requires complex engineering and computer analysis to develop a "programmed" crush of the engine compartment that in turn provides a minimum "controlled" deceleration of the passenger compartment. Further improvements are achieved by isolating front-end structures from the passenger compartment to minimize intrusion or deformation around the occupant. This method of controlling the "first impact," or the crash deceleration of the vehicle, is an important part of the total occupant protection system in current automobiles. Because of the greater crushing distance available in the front as compared to the side of a vehicle, however, this approach is much more effective in frontal than in lateral impacts.

FRIENDLY INTERIORS

For an unrestrained occupant, the controlled deceleration of the vehicle in

a frontal crash is followed by impact of the occupant against the vehicle interior (Figures 7*b* and 8*a*). During this “second impact,” the unbelted occupant continues to travel forward at the vehicle’s precrash velocity and strikes the interior, which has now come to rest in front of the occupant. Postcrash observations during the 1960s indicated that many injuries could be eliminated merely by removing hard knobs and sharp edges that occupants tended to hit, just as rigid hood ornaments have been removed for the benefit of pedestrians. Further protection of the occupant is achieved, however, by the use of energy-absorbing interior structures and load-distributing surfaces that minimize the occupant’s impact acceleration while spreading the remaining forces over a broader portion of the body’s strongest parts. This approach is often referred to as the “friendly” interior.

The concept of impact energy absorption has two aspects. First, the change in velocity experienced by the occupant must be extended over as long a time as possible. This can be done by having the occupant hit something that will deform in the direction of impact in a controlled manner, thus increasing the body’s stopping distance. Second, it is important that the yielding structure not spring back at the occupant, but rather deform permanently or recover only very slowly. Otherwise, the impact energy would be returned to the occupant and not absorbed by the structure.

The simplest method to achieve both energy absorption and load distribution is to install thick, slow-recovery foam padding wherever possible around the occupant. The use of padding, however, has practical limitations as well as limits on its effectiveness in severe crashes. As a result, other more complex systems have been developed to deal with particular occupant needs. Improved protection for the driver has been achieved by the development of an energy-absorbing steering system, which uses a force-limiting column to safely decelerate the driver. Another effective safety feature is the high-penetration-resistant windshield, which uses a stretchable plastic layer between two sheets of glass for head impact protection.

Energy-absorbing steering system. Identification of the need for an energy-absorbing steering system resulted from research on the sources of driver thoracic injury and on methods to decelerate an unrestrained body safely as it contacts the steering wheel. The basic concept was to design a steering column that would crush at a prescribed load, which was in turn not great enough to cause significant rib fracture. This device would increase the driver’s stopping distance, thus decreasing thoracic deceleration, and absorb impact energy. As indicated earlier, however, early development of the system was delayed by a lack of knowledge about the tolerance of the thorax

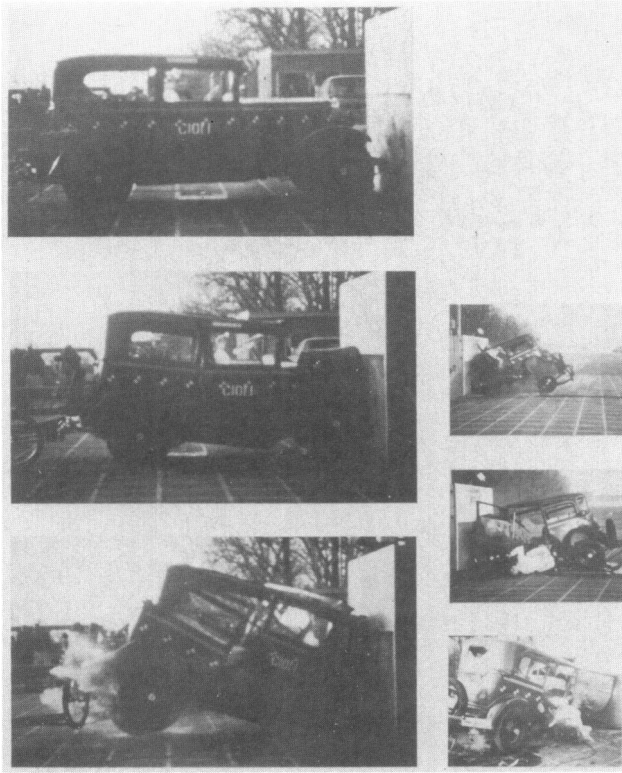


Fig. 7a. Sequence photos from a high-speed movie of a 1929 Chevrolet in a 50 kph (30 mph) frontal barrier crash with unrestrained dummies in the driver and right-front passenger seating positions.

to impact force. The cadaver experiments conducted in support of this project identified for the first time the force level needed for compression of the steering column that would minimize the risk of chest injury.¹⁰ Later tests demonstrated the need for load sharing between the chest and shoulders, accomplished through load distribution over the rim, spoke, and hub surfaces of the steering wheel.¹¹ Biomechanics research thus played an essential role in the development of this very effective occupant protection technology.

The energy-absorbing steering system was introduced in 1967 model vehicles. The final system (Figure 9) included a compressible ball-sleeve column, a steering wheel with improved load distribution and stiffness, and an anti-intrusion mounting bracket to reduce rearward motion of the steering system resulting from crush of the engine compartment.²⁵ When the load of the driver on the steering wheel exceeds the compressive force of the energy-

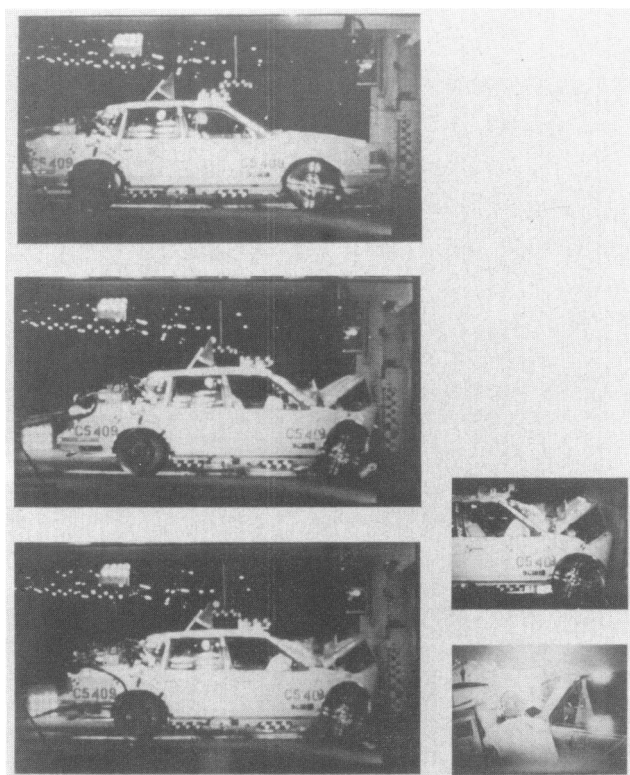


Fig. 7b. Sequence photos from a high-speed movie of a 1982 vehicle in a 50 kph (mph) frontal barrier crash with unrestrained dummies in the driver and right-front passenger seating positions.

absorbing element, the column slips out of the shear capsule, compresses, and absorbs energy (Figure 9). This system has proved effective in saving lives and reducing injuries. An evaluation by the National Highway Traffic Safety Administration²⁶ found that the overall risk of driver fatality in a frontal crash had been reduced by 12% since the introduction of the energy-absorbing steering system, but that the risk of serious injury (including death) due specifically to contact with the steering assembly had been reduced by 38%.

High-penetration-resistant windshields. Injury research during the early 1960s indicated that the windshield glass in use at the time caused significant facial laceration. These windshields were constructed of two glass layers with a thin (0.38 mm or 0.15 in) layer of plastic tightly bonded between them. This laminated glass was thus fairly brittle and would break and be penetrated

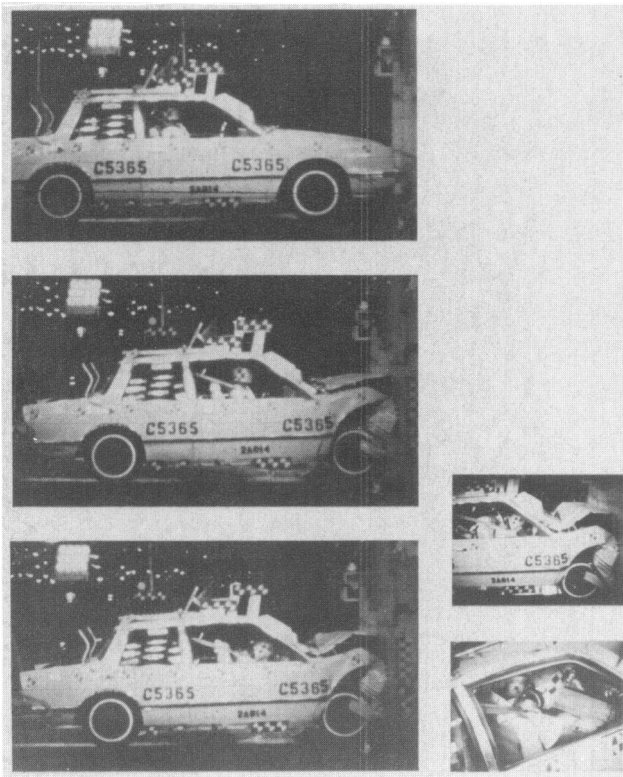


Fig. 7c. Sequence photos from a high-speed movie of a 1982 vehicle in a 50 kph (30 mph) frontal barrier crash with lap-shoulder belt restrained dummies in the driver and right-front passenger seating positions.

by the head in severe crashes (Figure 10), and often the face was raked against the jagged edge of the hole made by the head. It was determined that significant occupant protection could be achieved if the head could be kept from passing through the glass during impact while at the same time ensuring that the head would be safely decelerated to protect against concussion injury.

Extensive collaboration between engineering and medical experts was required to develop a laminated glass that would yield under impact, to increase the head's stopping distance, and still resist head penetration at higher impact speeds. The collaboration resulted in Gurdjian and Lissner conducting many series of head impact experiments at Wayne State University.²⁷ Their data were analyzed by Gadd,²⁸ who developed a weighted impulse criterion based on average head acceleration raised to the 2.5 power and impact duration to assess concussion injury risk. This so-called Gadd Severity Index (GSI) became a widely accepted method of head injury assess-

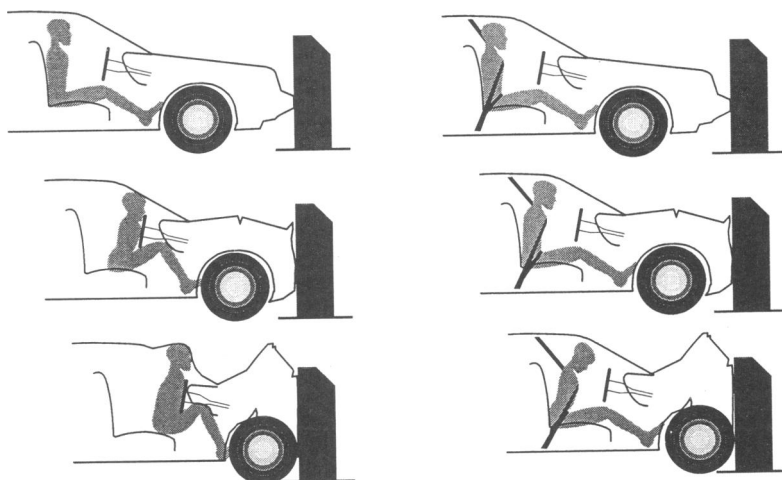


Fig. 8. Representation of occupant dynamics in a frontal barrier crash with unrestrained driver impacting the windshield, steering wheel, and instrument panel during the "second" impact and with a lap-shoulder belted driver "riding down" the vehicle deceleration and experiencing restraint from the "second" impact.

ment in anthropomorphic dummy tests and was the forerunner of the current Head Injury Criterion (HIC). These criteria are based on the principle that rigid body mechanics reasonably applies to the closed head injury problem so that head acceleration is a meaningful measure of brain injury risk.

Other research²⁹ led to the development of a chamois covering for the dummy head which provided an objective indication of the laceration protection of prototype windshields. Eventually, a series of a cadaver impact experiments was conducted using various prototype windshields in simulated vehicle crashes. These tests showed that a thicker (0.76 mm or 0.30 in) plastic inner-layer bonded more loosely to the two outer sheets of glass could provide a stretchable structure (Figure 10) with greater energy-absorbing capability that could still safely keep the head from penetrating the windshield at impact speeds up to about 29 mph.³⁰

The optimal characteristics for occupant protection and manufacturing feasibility were worked out in a joint effort between the automobile industry and the glass manufacturers, so that it was possible to introduce the new windshields in all 1967 model vehicles. Since then, high-penetration-resistant windshields have proved remarkably effective in reducing injuries to the face while not increasing the risk of brain concussion.³¹ A recent evaluation by the National Highway Traffic Safety Administration³² found a 70% reduc-

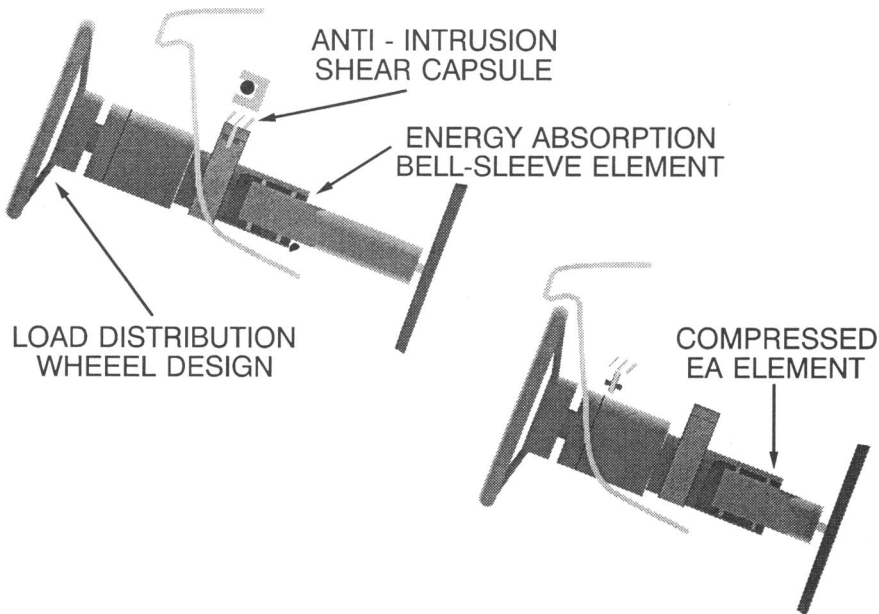


Fig. 9. Energy-absorbing steering system designed with a compressible element that resists driver impact and gradually deforms under load to protect an unrestrained driver. The system is shown in normal configuration and after full compression. The anti-intrusion shear capsule resists rearward movement of the steering system by deformations of the engine compartment.

tion in nonminor facial lacerations and fractures through the use of these windshields. More recent safety developments of windshield glass have focused on antilaceration inner shields, where a layer of plastic lines the inner surface of the windshield (Figure 10) further to prevent laceration of the face and scalp,^{33,34} and on a better method to assess head dynamics and facial contact force during glass impact.³⁵

RESTRAINT SYSTEMS

Although interior safety in the form of energy-absorbing structures and load-distributing surfaces has achieved tremendous gains in occupant crash protection, the crush distance available in even the friendliest interior is only a fraction of that needed to achieve safe occupant decelerations in a high-speed vehicle crash. Not surprisingly, the risk of serious injury and fatality increases uniformly with crash severity, as measured by vehicle velocity change (ΔV) (Figure 11). This risk is quite low in the least severe crashes but approaches 50% for serious injury and 25% for death of unrestrained occupants in accidents with a velocity change of more than 35 mph. This high risk

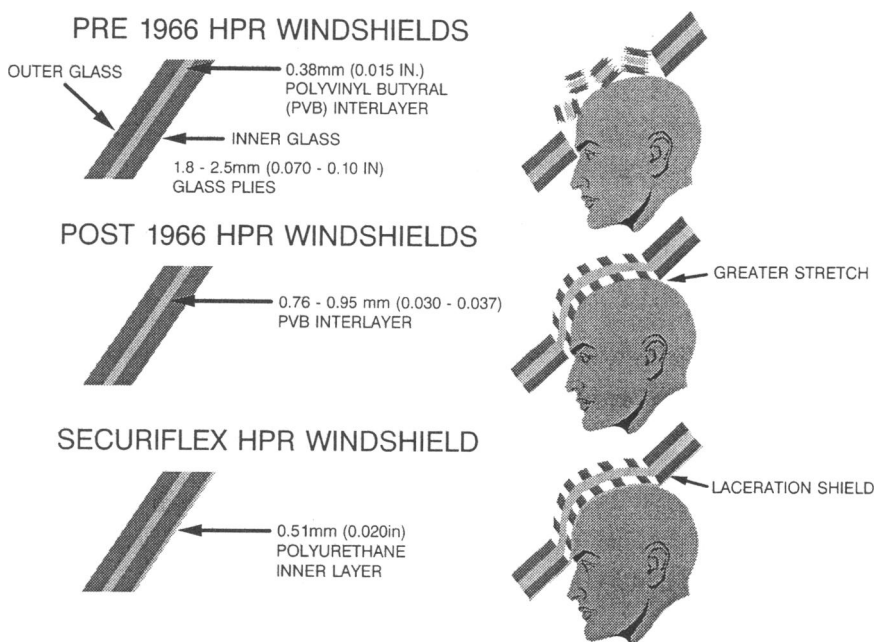


Fig. 10. High-penetration-resistant windshields which cushion the head during impact by stretch of the plastic interlayer and shield the face from laceration.

in the more severe crashes demonstrates the limits of protection possible with friendly interiors alone. In contrast, a similar plot of serious injury risk (Figure 11) for those using the available lap/shoulder belt restraint system is significantly lower for all but the highest crash speeds. These data indicate that further enhancement of occupant protection can only be achieved with restraint systems that allow the occupant to take better advantage of the vehicle's crashworthy structure, as described below.

Seat belt restraint. A snug-fitting lap/shoulder belt ties the occupant directly to the passenger compartment and allows that occupant to "ride-down" the crash as the vehicle's front end crushes (Figures 7c and 8b). This coupling and ride-down decelerates the occupant more gradually than current energy-absorbing interior structures can and eliminates the more severe occupant-to-interior "second collisions," provided the belts are themselves fairly tight. Belts are also designed to distribute restraining loads over strong skeletal structures, including the shoulder, rib cage, and pelvis, to optimize protection during deceleration. Finally, belts provide significant control over the occupant's motions during extreme impact, ensuring that the friendly inte-

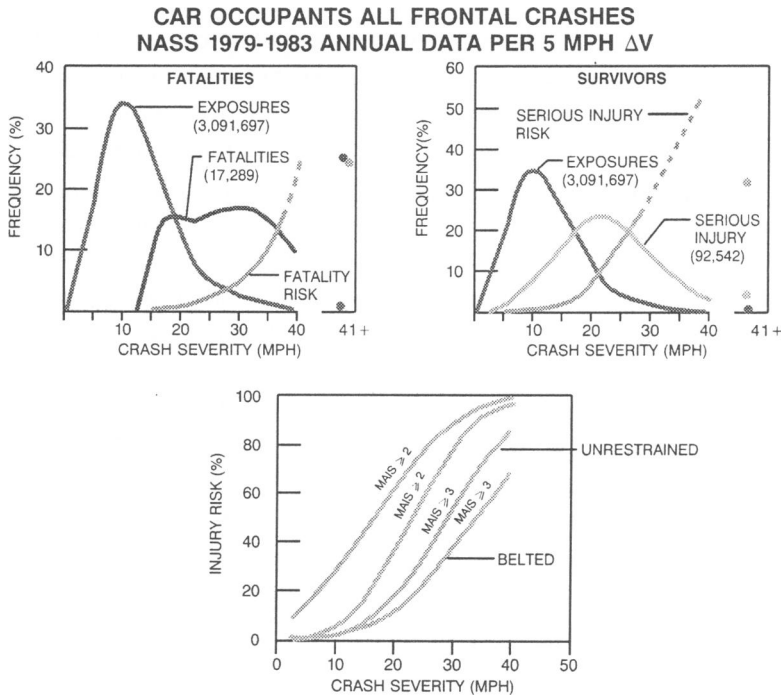


Fig. 11. Risk and distribution of serious injury and fatality as a function of the severity of the crash measured by the change in velocity of the struck vehicle (1979–82 NASS data)⁵² and comparative risk of injury for unrestrained and belted front seat occupants.

riors also load the body as designed. In contrast, the kinematics of unrestrained occupants and resulting interior contacts are highly unpredictable over the wide range of real world crashes. Even in rollover crashes, use of a lap/shoulder belt virtually eliminates the risk of ejection as well as the potential for disabling spinal cord injury by keeping the occupant in the seat.³⁶

A lap/shoulder belt restraint system adds significantly to the effectiveness of the total occupant protection system. When used, seat belts reduce the risk of death by 43%³⁷ and serious injury by 40%–70% in motor vehicle crashes.^{38,39}

Inflatable restraints. Air bag restraint systems were developed to overcome the primary weakness of belt systems: to be effective belts must be fastened in advance, usually by the occupant. Using a pyrotechnic device to generate nitrogen gas, a bag can be rapidly inflated during the early phase of vehicle frontal crush without action by the occupant. The bag then “fills” some of the space between the occupant and the interior, which couples the

occupant to the passenger compartment and achieves the safety benefits of ride-down and load distribution.⁴⁰ This coupling is only temporary, however, because the bag must be vented and deflate, so that it will not act as a spring. However, the rapid deployment speed of an air bag can present a risk to occupants, including children, who may be close to the bag during inflation. Its design requires a trade-off between a long inflation time to reduce the risk of inflation injury and a rapid inflation, quickly to fill the space between the occupant and the interior.⁴¹

Because air bags neither remain inflated nor provide lateral restraint, seat belts are needed adequately to control occupant kinematics over the range of crash types, including rollovers and side impacts. The current safety thinking, therefore, is to use inflatable restraints as a supplement to seat belts. The lap/shoulder belts would provide the primary coupling to the vehicle and control of kinematics, while the air bag would provide the additional protection of load distribution and crash energy absorption in the more severe frontal crashes. This combination of safety technologies can work with crash-worthy vehicles and friendly interiors further to enhance occupant protection.

COMPLEXITIES AND LIMITATIONS OF CRASH PROTECTION

Even with the advances that have been made in occupant impact protection, there remain problems difficult to resolve and some for which there are no solutions. Specifically, energy-absorbing structures that are evaluated only under high-severity test conditions, while effectively reducing injury in high-speed impacts, may actually increase injury in the more frequent low-speed impacts.⁴² It must also be recognized that, after a certain point, the capabilities of protective systems are exceeded and that the only way to protect against injury in very high severity crashes may be to avoid such crashes altogether.

The energy-absorbing steering system presents the apparent design dilemma indicated above. If the unrestrained driver's chest hits the system with maximum tolerable force, as in a high-speed crash, it would be advantageous if the structure were at maximum stiffness to provide controlled crush and energy absorption without bottoming out to a more rigid structure. Although some injury, such as rib fracture, might result, the system would give the driver a level of protection far beyond that possible with merely a padded surface. Such severe crashes, however, occur much less frequently than low-speed impacts, in which a driver would be better served by a "softer" steering column that would give way with very little force, so that no fractures or bruising would occur.

Thus we have a situation in which a system that was tested only at high speeds might reduce serious injuries in severe crashes while inadvertently generating minor injuries in low-speed impacts. If a 50% injury reduction among 100 high-severity crashes is offset by a 5% increase in injury among 1,000 low-severity crashes, there is no net gain in safety. Actual crash data indicate that the level of "harm" (defined as the sum of all injuries weighted according to their societal cost) associated with steering system and interior contact is fairly uniformly distributed across the range of frontal crash severities.^{43,44} Further improvements in this or any protective system would only be acceptable if injury were reduced uniformly for all crash configurations and severities or, alternatively, for a limited range of severity, as long as injury risks were not increased for other types of crashes.

In the case of the energy-absorbing steering system and other interior contact surfaces, an improvement has been the incorporation of graduated force level⁴²⁻⁴⁵ where proper evaluation of the design change required a broad program of crash investigation and laboratory testing using the most sophisticated test equipment and biomechanical knowledge to interpret the results. Merely testing occupant protection systems in government-mandated 30-mph frontal barrier collisions or single high-speed impact may not provide adequate information to judge their safety performance in the real world.

Unfortunately, there are upper limits on the ability of any combination of automobile crashworthiness, friendly interiors, and restraint systems to protect against very high-speed impacts, which result in severe passenger compartment deformation and high occupant accelerations. A detailed evaluation of 101 deaths of front-seat occupants, only four of whom were wearing belts, determined that half the victims could not have survived with any restraint system, including lap/shoulder belts or air bags.⁴⁶ A passenger vehicle has only so much space to crush, and humans have only so much tolerance to acceleration and direct impact. When these limits are exceeded, the result is serious or fatal injury. It is not realistic, for instance, to expect occupants to survive in the most severe real world collisions, which may involve very high speed impacts with heavy trucks or immovable objects.

The recent campaigns to encourage seat belt use, along with the installation of inflatable restraints, may unfortunately raise the public's expectations too high as to the potential safety benefits of restraint systems. Even the most sophisticated system cannot guarantee absolute protection from trauma. It cannot be denied, however, that vast improvements in occupant protection have been made during the last 20 years, and that the best strategy for anyone traveling in a motor vehicle is to take maximum advantage of the available

systems. At the same time, it must be recognized that, just as there is no universal inoculation against disease, there is no single solution to occupant protection. Vehicle-based systems are not enough and must be assisted by changes in the driving environment and in the behavior of drivers themselves.

Crash Injury in Society

Crash injury in the United States is a significant health issue with both immediate and long-term consequences for individuals and society.⁴⁷ Accidents of all types are the fourth leading cause of death, accounting for 5.5% of deaths in this country, and over half of these are from injuries sustained in motor-vehicle related crashes (Figure 12a). Accidents primarily afflict the young, accounting for nearly 55% of deaths of people 15 to 24 years old, and they are the leading cause of death for people under the age of 45.

Half the motor vehicle crash fatalities are occupants of passenger cars, but more than a quarter are pedestrians and motorcyclists, who are the least protected road users (Table I). In contrast, occupants of heavy vehicles, such as large trucks, have the lowest risk of death. The circumstances of fatal crashes are virtually limitless and range from low to high severities, but nearly 40% of fatal motor vehicle crashes involve drivers younger than 25 years old. Alcohol is also a significant factor in fatal crashes. Police and autopsy records show that at least one of the drivers had been drinking prior to about 50% of these crashes (Table II),⁴⁸ and medical research indicates that alcohol in body tissues increases their susceptibility to injury.^{49,50}

Looking at the problem from a different perspective, accidents are responsible for more years of lost productive life than any other cause (Figure 12b).⁵¹ These estimates take into account the age of the victim before age 65 and thereby ascribe the greatest loss to the youngest victims. Loss of life, however, is only part of the consequences of crash injury. In 1982 more than three million survivors of motor vehicle crashes were hospitalized for injuries; nearly 140,000 seriously injured people required an average of 12 days of hospital care and missed an average of 31 days of work.⁵² This represents a significant cost to society.

LONG-TERM CONSEQUENCES

Injury takes thousands of lives and injures millions of people annually. Although most of the injured recover, tens of thousands are seriously crippled and disabled.⁵³ Severe disabling injury from motor vehicle crashes has become a major health care issue. Most such disability is from brain and spinal

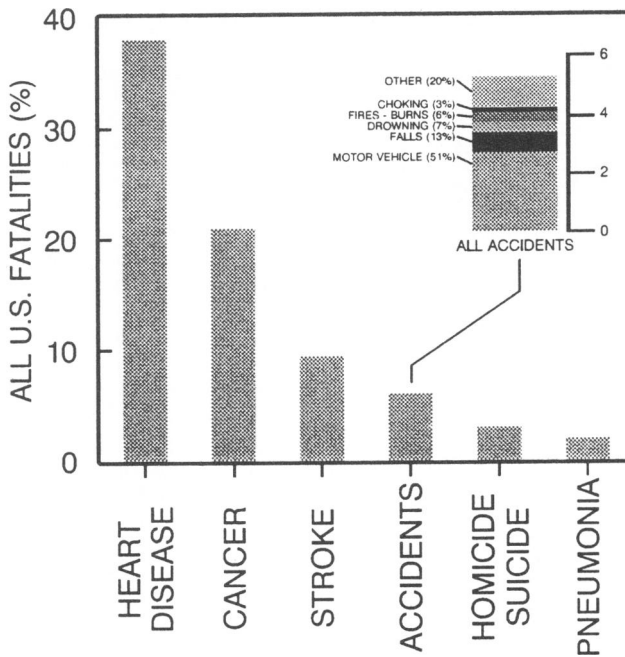


Fig. 12. Most frequent causes of U.S. fatalities and lost years of life before age 65 showing the importance of accidental causes (redrawn from!).

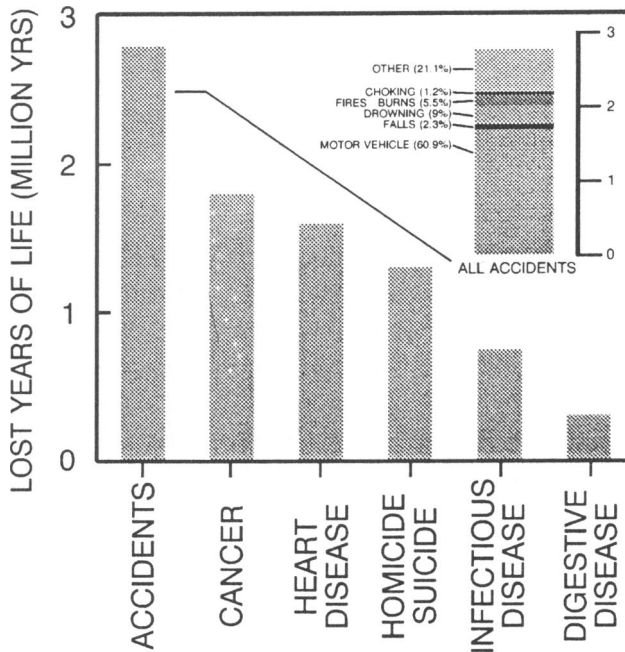


TABLE I. PEOPLE INVOLVED, PEOPLE INJURED, AND FATALITIES PER YEAR
IN MOTOR VEHICLE RELATED CRASHES, AVERAGE OF 1982–1985;
NATIONAL PROJECTION OF NASS; FATALITIES FROM FARS;

<i>Vehicle occupants</i>	<i>All involved Count</i>	<i>All injured Count</i>	<i>%</i>	<i>Seriously injured</i>		<i>Fatalities</i>	
				<i>Count</i>	<i>%</i>	<i>Count</i>	<i>%</i>
All cars	11,488,007	2,471,578	21.5	105,876	0.9	23,061	0.2
Towaway Cars	3,230,795	1,620,865	50.2	102,453	3.2	22,913	0.7
Non-towaway cars	8,257,212	850,713	10.3	3,423	0.0	148	0.0
Light trks & vans	2,633,781	446,098	16.9	28,919	1.1	6,545	0.2
Medium & hvy trks	470,249	40,554	8.6	3,393	0.7	1,067	0.2
Motorcycles	189,626	149,598	78.9	35,686	18.8	4,467	2.4
Buses	311,310	12,660	4.1	48	0.0	48	0.0
Other	12,398	1,805	14.6	665	5.4	244	2.0
<i>Non-Occupants</i>							
Pedestrians	125,733	105,779	84.1	26,980	21.5	6,998	5.6
Bicyclists	89,729	82,547	92.0	7,908	8.8	850	0.9
Other	51,193	16,269	31.8	699	1.4	201	0.4
Total	15,372,026	3,326,888	21.6	210,164	1.4	43,481	0.3

All injured: irrespective of severity or outcome
Seriously injured: at max AIS of 3 or higher, irrespective of outcome
Note: Shown counts and percentages are not mutually exclusive.

cord injuries, which permanently destroy motor, sensory, or cognitive function.

A typical crash victim receives multiple injuries.⁴⁷ The most frequent injuries are to the long bones and joints of the extremities. Although such injuries can involve extended temporary disability, they are generally limited in severity to lacerations and fractures, and the victim has a good chance of full recovery. The head is the second likeliest site of injury but receives the greatest number of critical injuries. In half of all victims the head is the most severely-injured region of the body, and many of these critical injuries, as well as a small fraction of the less severe ones, are not reversible.

Short duration unconsciousness is the most frequent brain injury and is generally thought to be recoverable, but evidence is mounting that permanent changes in function occur in some cases.⁵⁴ Longer duration concussion and coma represent more severe brain injuries and frequently have concomitant focal contusion of brain tissue. Although surgery can often preserve life, recovery of brain function is uncertain. Prompt and appropriate treatment of a person with injury to the central nervous system is also important to eventual recovery. There is often a “window of time” within which stabilization of

TABLE II. ALCOHOL INVOLVEMENT IN MOTOR VEHICLE CRASHES
1985 SUMMARY

<i>Crashes</i>	<i>Percent</i>	<i>Number</i>
All fatal (43,795 from FARS)	51%	22,360
Injury (3,365,000 from NASS)	16%	541,000
Property damage (17,100,000 from NASS)	8%	1,368,000
All crashes (19,300,000 from NASS)	11%	2,123,000

FARS—Fatal Accident Reporting System

NASS—National Accident Sampling System

the patient may halt metabolic changes that contribute to permanent tissue damage.

A recent study of hospital admission and discharge information⁵⁵ estimated an annual incidence of 204,000 people with acute brain trauma from transport related accidents (Figure 13), of which nearly half (48%) occur to vehicle occupants in crashes. Hospital release data⁵⁶ project that 17% of the total, or 34,200 people, will die annually, more than two thirds of these at the scene of the injury and the rest after admission to the hospital. The vast majority of survivors experience good recovery or only mild impairment upon release from the hospital, but 4% of the patients have moderate to severe impairment or remain in a vegetative state. Overall, 7% of all brain-injured patients discharged have a neurologic deficit or disability, as identified by a physician after the course of primary care. This translates to a national projection of 11,900 people annually with long-term neurologic sequelae from acute brain injury in transport accidents. Recent studies show that an additional 15,800 people, or 10% of those classified as having good recovery, may still experience psychological or cognitive deficits that can affect employment and life style.

Since central nervous system tissue is susceptible to irreversible damage, there is also concern that neck injuries may include the spinal cord, with possible loss of motor and sensory function. The annual incidence of transport related spinal cord trauma is estimated at 7,250 (Figure 13), more than half (58%) occurring to occupants of vehicle crashes.⁵⁷ The fatality rate for those with spinal cord injury in the United States is 47.6%, or 3,450 people annually. Nearly 75% of these die at the injury scene and the rest after hospital admission. Although patients admitted to the hospital with spinal cord injury appear to have a higher risk of dying in the hospital than those admitted with acute brain injuries (16% versus 6%), this difference merely reflects the large number of admissions for minor head injury. In contrast to acute brain injury, the vast majority of the 3,770 survivors experience impair-

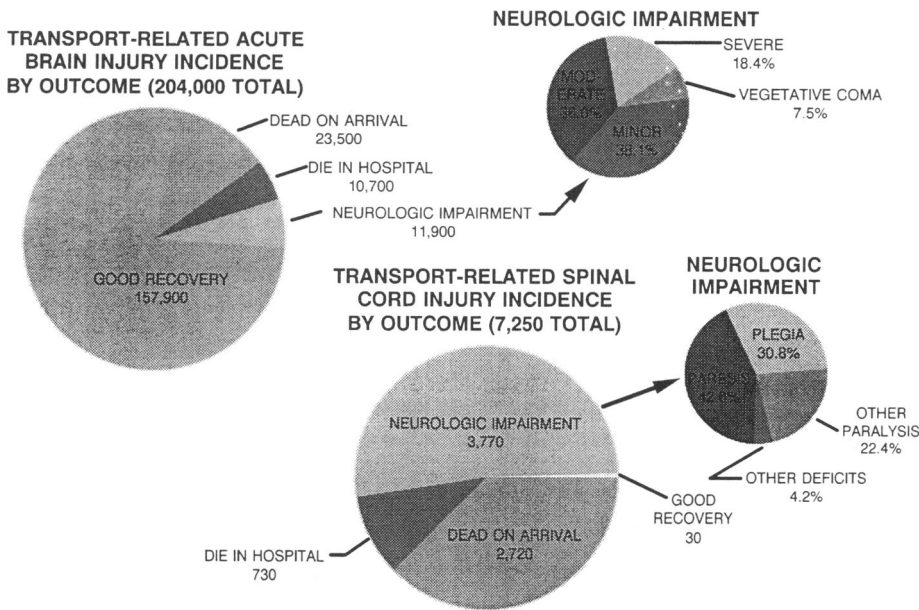


Fig. 13. Projected incidence of transport related acute brain and spinal cord injury including neurologic impairment at the time of hospital discharge (drawn from data in 55-57).

ment at the time of discharge. Some are quadriplegic (4.2%), many are paraplegic (26.4%) or otherwise paralyzed (22.2%), others experience paresis or muscle weakness (42.4%), and 4.2% have other minor deficits. Although 44% of the patients admitted with quadriplegia or paraplegia improve and are released with a less severe impairment, fewer than 1% of all admissions are released with good recovery.

The foregoing documents that brain and spinal cord injuries have clear risks of permanent disability and fatality. Further, recent analysis of injury patterns in Europe indicates that brain and spinal cord injuries continue to be an important accident consequence, particularly for the driver, even with widespread safety belt use.³⁹ Although hospital admission data indicate that brain injury is 28 times more frequent than spinal cord injury, when outcome is considered, the incidence of permanent disability from brain injury is approximately three times that of spinal cord injury, seven times if mild brain injury disability from the “good” recovery group is included. Figure 14 shows the expected national incidence of permanent central nervous system disability to occupants injured in motor vehicle crashes. When permanent paralysis from spinal cord injury is compared with the severe and vegetative

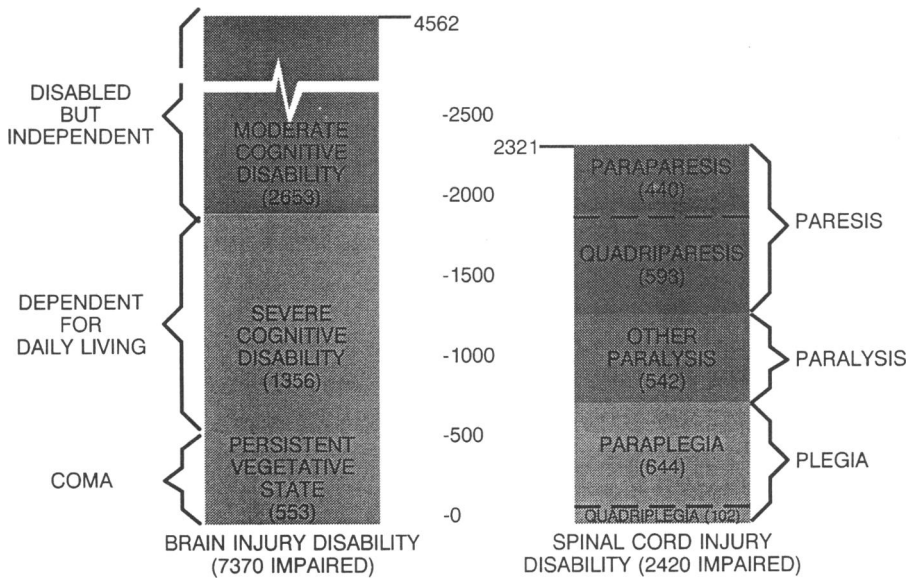


Fig. 14. Projected incidence of disabling brain and spinal cord injury of passenger car occupants (drawn from data in 55-57).

brain injury cases, spinal cord paralysis is nearly as frequent as severe cognitive or functional brain injury, actually more than 30% greater if quadriplegia/paraplegia is compared with vegetative coma. The severely impaired victims require attendant care in nearly half of the cases.⁵⁸ Only one in 10 of these victims returns to gainful employment, and nearly 40% are unemployable for years after the injury. These individuals face an average life expectancy of 36 years, and about a quarter live 50 years or more after the crash injury. Thus, the expected national prevalence of the most severe central nervous system disability from vehicle occupant injury is approximately 115,000 (nearly 225,000 for transport-related cases).

A NATIONAL STRATEGY

Crash injury costs society billions of dollars annually, a recent estimate by the National Highway Traffic Safety Administration approaching \$60 billion.⁵⁹ Much of the human damage, work loss, and disability from accidents may be preventable by more effective accident preventive measures, occupant protection technologies, and medical treatment and rehabilitation; yet trauma prevention research receives only a small fraction of federal research dollars invested in health problems.¹ This is likely to change as both the

public and the various professionals working in related fields understand and recognize that there is a national problem, it can be controlled, although not eliminated, and the solutions are complex and will involve contributions from and cooperation among a wide range of groups.

The public has generally had a low perception of the injury risk involved in highway vehicle travel. This perception is reinforced by the millions of miles of accident-free travel that occur each day. There are also, however, thousands of crashes each day and deaths each year. Better awareness of this risk should encourage individuals to use the available belt restraint systems, which enhance the built-in protection in automobiles, as the best defense against the drunk or careless driver. We are currently seeing an increase in the use of lap/shoulder and lap-only belt restraint systems, because of the adoption of mandatory belt use laws in many states and the demonstrated effectiveness of child safety seat use.⁶⁰ This change is causing a shift in the patterns of injuries and disabilities in motor vehicle crashes, including not only decreases in severe head and chest injuries but an increase in belt-related injuries in the more severe crashes. Such shifts require awareness and training among emergency medical personnel to look for injuries that may be unfamiliar and not immediately apparent. They also demand the collection of more complete biomechanical response and tolerance data so that protective systems can be further improved for a wider range of crash situations. Although we have made progress, the preceding review of the crash injury situation in America demonstrates how far we yet have to go in preventing and controlling injury to the motoring public.

Injury control is a complicated problem requiring the cooperation and consensus of many groups who are most affected. These include the automotive industry, insurance industry, federal transportation agencies, public health agencies, advocacy groups for consumers and injured persons, injury research organizations, and other academic and public groups with a voice and interest in reducing motor vehicle crash injuries, disabilities, and fatalities. The problem of injury in America can be controlled, but resources are limited and their effective use is critical. It may be, for instance, that vehicle-based crash protective systems have reached a plateau of effectiveness, and that significant further improvements will only come with very large expenditures. The same resources applied to driver behavior or the road environment may result in a larger payoff, but decisions regarding safety investment cannot be made by a single group or organization.

Safety experts agree that there is no single solution to the occupant protection problem, and that a combination of technologies will be the most effec-

tive defense against the wide range of real-world crash situations. Likewise, a combination of approaches is required to control the entire chain of events of accident, injury, and long-term consequences. This calls for a national strategy, arrived at by consensus among entities that may formerly have been at odds, and supported by quality engineering, medical, and social research.

Summary

Trauma, with particular emphasis on blunt impact and acceleration injury in the automotive environment, is compared to major diseases with regard to its characteristics and effects on individuals and society. The causes of motor vehicle crashes are reviewed and evidence is presented that the vehicle is a minor factor in accidents. Injury causation is discussed in engineering terms, and several approaches are presented for setting criteria to establish injury thresholds and to evaluate the effectiveness of injury preventive systems. The history and current status of occupant protection technologies are described, including vehicle crashworthiness, friendly interiors, and restraint systems, and special attention is given to the need to use various systems in combination for maximum effectiveness. Finally, the broad scope of trauma's impact on society is described in terms of long-term consequences, including human disability and loss of productivity, and an appeal is made for a balanced and cooperative approach among various organizations and interests further to control automotive impact trauma in the future.

REFERENCES

1. Committee on Trauma Research, Commission on Life Sciences, Nat. Research Council and the Institute of Medicine: *Injury in America—A Continuing Public Health Problem*. Washington, D.C., National Academy Press, 1985.
2. *Stedman's Medical Dictionary*, 24th edition. Baltimore, MD, Williams and Wilkins, 1982.
3. Treat, J.R.: A study of precrash factors involved in traffic accidents. *HSRI Res. Rev.* 10(6)/11(1):1-35, 1980.
4. Viano, D.C., King, A.I., Melvin, J.W., and Weber, K.: *Injury Biomechanics Research: An Essential Element in the Prevention of Trauma*. General Motors Research Laboratories Publication GMR-4951, February 1985. *J. Biomech.* In press.
5. Ommaya, A.K.: Biomechanics of Head Injury: Experimental Aspects. In: *The Biomechanics of Trauma*, Nahum, A.M. and Melvin, J.W., editors. Norwalk, Conn., Appleton-Century-Crofts, 1985, pp. 245-70.
6. Lau, I.V., Horsch, J.D., Viano, D.C., and Andrzejak, D.V.: Biomechanics of liver injury by steering wheel loading. *J. Trauma* 21:1-11, 1987.
7. Kroell, C.K., Gadd, C.W., and Schneider, D.C.: Biomechanics in Crash Injury Research. *19th International ISA Aerospace Instrumentation Symposium*. Las Vegas, May 1973.
8. Stapp, J.P.: Voluntary Human Tolerance Levels. In: *Impact Injury and Crash Protection*, Gurdjian, E.S., Lange, W.A., Patrick, L.M., and

- Thomas, L.M., editors. Springfield, Thomas, 1970, pp. 308–49.
9. Eiband, A.M.: Human Tolerance to Rapidly Applied Acceleration: A Survey of the Literature. NASA Memo No. 5–19–59E. Washington, D.C., Nat. Aeronautics and Space Administration, 1959.
 10. Patrick, L.M., Kroell, C.K., and Mertz, H.J.: Forces on the Human body in Simulated Crashes. *Proceedings of the Ninth Stapp Car Crash Conference*. Minneapolis, October 1965, pp. 237–60.
 11. Gadd, C.W. and Patrick, L.M.: Systems Versus Laboratory Impact Tests for Estimating Injury Hazard. *Society of Automotive Engineers Automotive Engineering Congress*. Paper #680053. Detroit, January 1968.
 12. Kroell, C.K.: Thoracic Response to Blunt Frontal Loading. In: *The Human Thorax-Anatomy, Injury and Biomechanics*. SAE Publication P-67. Warrendale, PA, Society of Automotive Engineers, 1976, pp. 49–78.
 13. Viano, D.C.: Thoracic Injury Potential. *Proceedings of the Third Int. Meeting on the Simulation and Reconstruction of Impacts in Collisions*. Lyon, France, 1978, pp. 142–56.
 14. Neathery, R.F., Kroell, C.K., and Mertz, H.J.: Prediction of Thoracic Injury from Dummy Responses. *Proceedings of the Nineteenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #751151. San Diego, CA, November 1975, pp. 195–316.
 15. Foster, J.K., Kortge, J.O., and Wolanin, M.J.: Hybrid III—a Biomechanically-Based Crash Test Dummy. *Proceedings of the Twenty-First Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #770938. New Orleans, October 1977.
 16. Horsch, J.D. and Viano, D.C.: Influence of the Surrogate in Laboratory Evaluation of Energy-Absorbing Steering System. *Proceedings of the Twenty-Ninth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #841660. Washington, D.C., October 1985, pp. 261–74.
 17. Jonsson, A., Clemedson, C.J., Sundquist, A.B., and Arvebo, E.: Dynamic factors influencing the production of lung injury in rabbits subjected to blunt chest wall impact. *Aviat. Space Environ. Med.* 50:325–37, 1979.
 18. Lau, I.V. and Viano, D.C.: Influence of impact velocity on the severity of non-penetrating hepatic injury. *J. Trauma* 21:115–23, 1981.
 19. Lau, I.V., Viano, D.C., and Doty, D.B.: Experimental cardiac trauma-ballistics of a captive bolt pistol. *J. Trauma* 21:39–41, 1981.
 20. Stein, P.D., Sabbah, H.N., Viano, D.C., and Vostal, J.J.: Response of the heart to nonpenetrating cardiac trauma. *J. Trauma* 22:34–73, 1982.
 21. Viano, D.C. and Lau, I.V.: Role of impact velocity and chest compression in thoracic injury. *Aviat. Space Environ. Med.* 54:16–21, 1983.
 22. Viano, D.C. and Lau, I.V.: Thoracic Impact: A Viscous Tolerance Criterion. *Proceedings of the Tenth Experimental Safety Vehicle Conference*. Oxford, England, 1985, pp. 104–14.
 23. Horsch, J.D., Lau, I.V., Viano, D.C., and Andrzejak, D.V.: Mechanism of Abdominal Injury by Steering Wheel Loading. *Proceedings of the Twenty-Ninth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #851724. Washington, D.C., October 1985, pp. 69–78.
 24. Lau, I.V. and Viano, D.C.: The Viscous Criterion—Bases and Applications of an Injury Severity Index for Soft Tissues. *Proceedings of the Thirtieth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #861882. San Diego, October 1986, pp. 123–42.
 25. Marquis, D.P.: The General Motors Energy Absorbing Column. *Society of Automotive Engineering Congress*. Paper #670039. Detroit, January 1967.
 26. Kahane, C.J., *An Evaluation of Federal Motor Vehicle Safety Standards for Passenger Car Steering Assemblies*. DOT HS 805 705. Washington, D.C., National Highway Traffic Safety Administration, 1981.
 27. Gurdjian, E.S. Lissner, H.R., and Patrick, L.M.: Concussion-Mechanism

- and Pathology. *Proceedings of the Seventh Stapp Car Crash Conference, Society of Automotive Engineers*. Los Angeles, November 1963, pp. 470–83.
28. Gadd, C.W.: Use of a Weighted Impulse Criterion for Estimating Injury Hazard. *Proceedings of the Tenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #660793. Holloman Air Force Base, NM, November 1966, pp. 164–74.
 29. Pickard, J., Brereton, P., and Hewson, A.: An Objective Method of Assessing Laceration Damage to Simulated Facial Tissues—The Triplex Laceration Index. *Proceedings of the 17th American Association for Automotive Medicine Conference*. Oklahoma City, November 1973, pp. 148–65.
 30. Huelke, D.F., Grabb, W.C., Dingman, R.O., and Oneal, R.M.: The new automotive windshield and its effectiveness in reducing facial lacerations. *Plastic Reconstr. Surg.* 41:554–59, 1968.
 31. Reiser, R.G. and Chabal, J.: Safety Performance of Laminated Glass Structures. *Society of Automotive Engineers Midyear Meeting*. Paper #700481. Detroit, 1970.
 32. Kahane, C.J.: *An Evaluation of Windshield Glazing and Installation Methods for Passenger Cars*. DOT HS 806 693. Washington, D.C., National Highway Traffic Safety Administration, 1985.
 33. Jandeleit, O. and Orain, R.: Performance of a Non-Lacerative Windshield in Case of Accident and Under Environmental Conditions. Paper #770246. *SAE Trans.* 86:1101–14, 1977.
 34. Browne, A.L.: Dynamic Test performance of a Concept Two-Ply Windshield. Paper #861406. Warrendale, PA, Society of Automotive Engineers, 1986.
 35. Viano, D.C., Melvin, J.W., McCleary, J.D., et al.: Measurement of Head Dynamics and Facial Contact Forces in the Hybrid III Dummy. *Proceedings 30th Annual Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #861891. San Diego, 1986, pp. 269–90.
 36. Huelke, D.F., Lawson, T.E., Scott, R., and Marsh, IV, J.C.: The Effectiveness of Belt Systems in Frontal and Rollover Crashes. Paper #770148. *SAE Trans.* 86(7):626–34, 1977.
 37. Evans, L.: The effectiveness of safety belts in preventing fatalities. *Acc. Anal. Prev.* 18:229–41, 1986.
 38. *Advances in Belt Restraint Systems: Design, Performance and Usage*. Special Publication P-141. Warrendale, PA, Society of Automotive Engineers, 1984.
 39. Rutherford, W.H., Greenfield, T., Hayes, H.R.M., and Nelson, J.K.: *The Medical Effects of Seat Belt Legislation in the United Kingdom*. London, Her Majesty's Stat. Off., 1985.
 40. *Passenger Car Inflatable Restraint Systems: A Compendium of Published Safety Research*, Viano, D., editor. Publication PT-31. Warrendale, PA, Society of Automotive Engineers, 1987.
 41. Horsch, J.D. and Culver, C.C.: A Study of Driver Interaction with an Inflating Air Cushion. *Proceedings of the Twenty-Third Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #791029. San Diego, CA, 1979, pp. 799–823.
 42. Horsch, J.D.: Evaluation of Occupant Protection from Laboratory Tests. *Society of Automotive Engineers. Int. Congress and Exposition*. Paper #870222. Detroit, February 1987.
 43. Malliaris, A.C., Hitchcock, R., and Hedlund, J.: A Search for Priorities in Crash Protection. In: *Crash Protection. Society of Automotive Engineers, International Congress and Exposition*. Paper #820242. Detroit, February 1982, pp. 1–34.
 44. Viano, D.C.: An Evaluation of the Benefits of Energy-Absorbing Material in Side-Impact Protection. *Proceedings of the Thirty-First Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #872212. New Orleans, November 1987.
 45. Viano, D.C.: Further Evaluation of the Benefits of Energy-Absorbing Material in Side-Impact Protection: Constant Force vs Constant Stiffness Materials. Paper #871480. *Proceedings of the Thirty-First Stapp Car Crash Conference*.

- ence, *Society of Automotive Engineers*. Paper #871480. New Orleans, November 1987, pp. 205-24.
46. Huelke, D.F., Sherman, H.W., and Murphy, M.J.: Effectiveness of Current and Future Restraint Systems in Fatal and Serious Injury Automobile Crashes. *Society of Automotive Engineers. Int. Congress and Exposition*. Paper #790323. Detroit, February 1979.
 47. Automobile-related injuries. *J.A.M.A.* 249:3216-22, 1983.
 48. Alcohol and the driver. *J.A.M.A.* 255:522-27, 1986.
 49. Waller, P.F. Stewart, J.R., Hansen, A.R., et al.: The potentiating effects of alcohol on driver injury. *J.A.M.A.* 256:1461-66, 1986.
 50. Anderson, T.E. and Viano, D.C.: Effect of Acute Alcohol Intoxication on Injury Tolerance and Outcomes. In: *Alcohol, Drugs and Traffic Safety*, Noordzig, P. and Roszbach, R., editors. Elsevier, Amsterdam, Excerpta Medical Elsevier Sci. Pub., 1987, pp. 251-54.
 51. Perloff, J.D., LeBailly, S.A., Kletke, P.R., et al.: Premature death in the United States: Years of life lost and health priorities. *J. Public Health Pol.* 5:167-84, 1984.
 52. National Highway Traffic Safety Administration National Center for Statistics and Analysis: *Report on Traffic Accidents and Injuries, 1981: National Accident Sampling System*. DOT HS 806 530. 1983.
 53. Luchter, S.: Traffic Related Disabilities and Impairments and Their Economic Consequences. *Crash Injury Impairment and Disability: Long Term Effects. Society of Automotive Engineers Int. Congress and Exposition*. Paper #860505. Detroit, February 1986, pp. 93-114.
 54. Rimel, R., Giordani, B., Barth J., et al.: Disability caused by minor head injury. *Neurosurgery* 9:221-8, 1981.
 55. Anderson, T.E. and Viano, D.C.: Estimated United States Incidence of Disabling and Fatal Central Nervous System Traumas: A Need to Reevaluate Priorities? *Proceedings Int. IRCOB Conference on the Biomechanics of Impacts*. Zurich, September 1986, pp. 41-51.
 56. Kraus, J.F., Black, M.A., Hessol, N., et al.: The incidence of acute brain injury and serious impairment in a defined population. *Am. J. Epidemiol.* 119:186-201, 1984.
 57. Kraus, J.F.: Epidemiological Aspects of Acute Spinal Cord Injury: A Review of Incidence, Prevalence, Causes and Outcome. In: *Central Nervous System Trauma Status Report*, Becker, D.P. and Povlishock, J.T. editors. Bethesda, MD, Nat. Inst. of Neurological and Communicative Disorders and Stroke, 1985.
 58. Staggering cost of serious auto crashes continues. *J. Am. Ins.* 59:10-13, 1983.
 59. National Highway Traffic Safety Administration: *The Economic Cost to Society of Motor Vehicle Accidents*. DOT HS 806 342. Washington, D.C., 1983.
 60. Kahane, C.J.: *An Evaluation of Child Passenger Safety—The Effectiveness and Benefits of Safety Seats*. DOT HS 806 890. Washington, D.C., National Highway Traffic Safety Administration, 1986.
 61. U.S. Department of Health, Education, and Welfare: *Healthy People; Report to the Surgeon General on Health Promotion and Disease Prevention*. DHEW Publication No. (PHS) 79-55071. Washington, D.C., DHEW, 1979.
 62. National Research Council, Committee on Trauma and Committee on Shock: *Accidental Death and Disability: The Neglected Disease of Modern Society*. Washington, D.C., Nat. Research Council, 1966.
 63. Baker, S.P., O'Neill B., and Karpf, R.S.: *The Injury Fact Book*. Lexington, Ma, Lexington Books, 1984.
 64. Trunkey, D.D.: Trauma. Accidental and intentional injuries account for more years of life lost in the U.S. than cancer and heart disease. Among the prescribed remedies are improved preventive efforts, speedier surgery and further research. *Sci. Am.* 249:28-35, 1983.
 65. American Trauma Society: *The Need for a National Trauma Institute*. Chicago, American Trauma Soc., 1982.
 66. Stone, W.S.: Trauma: A continuing

- U.S. health problem. *J. Trauma* 17:89-92, 1977.
67. National Safety Council: Safety in the Transition. In: *Accident Facts*. Chicago, Nat. Safety Council, 1984, pp. 8,9.
 68. Waller, J.: *Injury Control: A Guide to Health and Safety Professionals to the Causes and Prevention of Trauma*. Lexington, MA, Lexington Books, 1984.
 69. Robertson, L.S.: *Injuries—Causes, Control Strategies, and Public Policy*. Lexington, MA, Lexington Books, 1983.
 70. Oestern, H.J., Trentz, O., Hempelmann, G., et al.: Cardiorespiratory and metabolic patterns in multiple trauma patients. *Resuscitation* 7:169-83, 1979.
 71. National Research Council, Committee on Emergency Medical Services: *Roles and Resources of Federal Agencies in Support of Comprehensive Emergency Medical Services*. DHEW Publication No. 73-13. Rockville, MD, DHEW, 1973.
 72. Gann, D.S. and Amaral, J.F.: Pathophysiology of Trauma and Shock. In: *Surgery of Trauma*, 4th ed., Zuidema, G.D., Rutherford, R. B., and Ballenger, W. F., editors, Philadelphia, Sanders, 1984.
 73. Toole, J.F. and Toole, W.W.: Federal funding for research in stroke and trauma—A clinical investigator's viewpoint. *Stroke* 15:168-71, 1984.
 74. DiMaio, V.J.: Penetration and perforation of skin by bullets and missiles. A review of the literature. *Am. J. Forens. Med. Pathol.* 2:107-10, 1981.
 75. Adams, D.B.: Wound ballistics: A review. *Milit. Med.* 147:831-35, 1982.
 76. Wolf, R.A.: Four facets of automotive crash injury research. *N.Y. State J. Med.* 66:1798-1813, 1966.
 77. Hartunian, N.S., Smart, C.N., and Thompson, M.S.: *The Incidence and Economic Costs of Major Health Impairments: A Comparative Analysis of Cancer, Motor Vehicle Injuries, Coronary Heart Disease, and Stroke*. Lexington, MA, Heath, 1981.
 78. Haddon, W., Jr., and Baker, S.P.: Injury Control. In: *Preventive and Community Medicine*, 2nd ed., Clark, D.W., and MacMahon B., editors. Boston, Little, Brown, 1981.
 79. Morse, T.S.: The American Trauma Society—Lessons learned from cancer and coronaries. *Surgery* 73:806-09, 1973.
 80. Kahane, C.J.: The National Highway Traffic Safety Administration's Evaluations of Federal Motor Vehicle Safety Standards. *Society of Automotive Engineers Government/Industry Meeting*. Paper #840902. Washington, D.C., May 1984.
 81. Hess, R.L.: Trends and factors in the evolution of traffic safety in the U.S. *UMTRI Res. Rev.* 14:January-February, 1984.
 82. Goldbaum, G.M., Remington, P.L., Powell, K.E., et al.: Failure to use seat belts in the United States. *J.A.M.A.* 255:2459-62, 1986.
 83. *Closing the Gap*. Health Policy Project Interim Summary, The Carter Center of Emory University Health Policy Consultation. November 1984.
 84. Eastman, J.W.: Styling vs Safety: The American Automobile Industry and the Development of Automotive Safety, 1900-1966. Ph.D. dissertation, University of Florida, 1973.
 85. DeHaven, H.: Beginnings of Crash Injury Research. *Proceedings of the Thirteenth Stapp Car Crash Conference, Society for Automotive Engineers*. Boston, December 1969, pp. 422-28.
 86. DeHaven, H.: Mechanics of injury under force conditions. *Mech. Eng.* 66:264-268, 1944.
 87. Research on crash injuries. Editorial. *J.A.M.A.*:524, 1946.
 88. Schwimmer, S. and Wolf, R.A.: *Leading Causes of Injury in Automobile Accidents*. Automotive Crash Injury Research of Cornell University, June 1982.
 89. Huelke, D.F. and Gikas, P.W.: How Do They Die? Medical-Engineering Data from On-Scene Investigations of Fatal Automobile Accidents. *International Automotive Engineering Congress*. Paper #1003A. Detroit, January 1965.

90. Foege, W.H., Amler, R.W., and White, C.C.: Closing the gap—Report of the Carter Center Health Policy Consultation. *J.A.M.A.* 254:1355–58, 1985.
91. Malliaris, A.C., Hitchcock, R., and Hansen, M.: Harm Causation and Ranking in Car Crashes. *Int. Congress and Exposition, Society of Automotive Engineers*. Paper #850090. Detroit, February, 1985.
92. United States Department of Transportation: *Head and Neck Injury Criteria—A Consensus Workshop*. DOT-HS-806-434. Washington, D.C., 1981.
93. Gurdjian, E.S. and Gurdjian, E.S.: Re-evaluation of the biomechanics of blunt impact injury of the head. *Surg. Gynecol. Obstet.* 140:845–50, 1961.
94. Gurdjian, E.S. and Gurdjian, E.S.: Cerebral contusions: Re-evaluation of the mechanism of their development. *J. Trauma*, 16:35–51, 1976.
95. Gurdjian, E.S.: *Impact Head Injury*. Springfield, IL, Thomas, 1975.
96. Courville, C.B.: The mechanism of coup-contrecoup injuries of the brain. *Bull. Los Angeles Neurol. Soc.* 15:72–86, 1950.
97. Ommaya, A., Grubb, R.L., and Naumann, R.A.: Coup and contrecoup injury: Observations on the mechanics of visible brain injuries in the rhesus monkey. *J. Neurosurg.* 35:503–16, 1971.
98. Gennarelli, T.A. and Thibault, L.E.: Biomechanics of acute subdural hematoma. *J. Trauma* 22:1982.
99. Gennarelli, T.A., Thibault, L.E., Adams, J.H., et al.: Diffuse axonal injury and traumatic coma in the primate. *Ann. Neurol.* 12:564–74, 1982.
100. Hess, R.L., Weber, K., and Melvin, J.W.: *Review of Literature and Regulation Relating to Head Impact Tolerance and Injury Criteria*. The University of Michigan Highway Safety Research Institute, UM-HSRI-80-52-1, 1980.
101. Patrick, L.M., Kroell, C.K., and Mertz, H.J.: Forces on the Human Body in Simulated Crashes. *Proceedings of the Ninth Stapp Car Crash Conference, Society of Automotive Engineers*. Minneapolis, October 1966, pp. 237–59.
102. Rimel, R., Giordani, B., Jeffrey, B., et al.: Moderate head injury: Completing the clinical spectrum of brain trauma. *Neurosurgery* 11:344–51, 1982.
103. Gennarelli, T.: Influence of the type of intracranial lesion on outcome from severe head injury. *J. Neurosurg.* 56:26–32, 1982.
104. Kraus, J.F., Black, M.A., Hessol, N., et al.: The incidence of acute brain injury and serious impairment in a defined population. *Am. J. Epidemiol.* 119:186–201, 1984.
105. Report on the National Head and Spinal Cord Injury Survey conducted for the National Institute of Neurological and Communicative Disorders and Stroke. *J. Neurosurg.* (Suppl.):S1–43, 1981.
106. Langfitt, T.W., Gennarelli, T.A., Obrist, W.D., et al.: Prospects for the future in diagnosis and management of head injury: Pathophysiology, brain imaging, and population-based studies. *Clin. Neurosurg.* 29:353–76, 1982.
107. Collins, W.F.: A review and update of experiment and clinical studies of spinal cord injury. *Paraplegia* 21:204–19, 1983.
108. Portnoy, H.D., McElhaney, J.H., and Melvin, J.W.: Mechanism of Cervical Spine Injury in Auto Accidents. *Proceedings of the 15th Annual Conference of the American Association for Automotive Medicine, Society for Automotive Engineers*. Colorado Springs, CO, October 1971. pp. 58–83.
109. Holdsworth, F.: Fractures, dislocations, and fracture—dislocations of the spine. *J. Bone Joint Surg.* 52A:1534–51, 1970.
110. Holdsworth, F.W.: Fractures, dislocations, and fracture-dislocation of the spine. *J. Bone Joint Surg.* 45B:6–20, 1963.
111. Schneider, R.C.: Cervical spine and spinal cord injuries. *Mich. Med.* 63:773–86, 1964.
112. Kazarian, L.: Injuries of the human spinal column: Biomechanics and injury classification. *Exercise Sport Sci. Rev.* 9:297–352, 1981.
113. Mertz, H.J., Neathery, R.F., and Culver, C.C.: Performance Requirements

- and Characteristics of Mechanical Necks. *Proceedings of the General Motors Symposium on Human Impact Response*. Warren, MI, October 1972.
114. Wismans, J. and Spenny, C.H.: Performance Requirements for Mechanical Necks in Lateral Flexion. *Proceedings of the Twenty-Seventh Stapp Car Crash Conference with the International Research Committee on Biokinetics of Impacts*. Paper #831613. San Diego, CA, October 1983, pp. 137-48.
 115. Mertz, H.J. and Patrick, L.M.: Strength and Response of the Human Neck. *Proceedings of the Fifteenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #710855. Los Angeles, November 1971.
 116. Mertz, H.J., Neathery, R.F., and Culver, C.C.: Performance Requirements and Characteristics of Mechanical Necks. In: *Human Impact Response-Measurement and Simulation*. New York, Plenum Press, 1973.
 117. Culver, C.C., Neathery, R.F., and Mertz, H.J.: Mechanical Necks with Human-like Response. *Proceedings of the Sixteenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #720959. Detroit, November 1972.
 118. Anderson, T.E.: Spinal cord contusion injury: Experimental dissociation of hemorrhagic necrosis and subacute loss of axonal conduction. *J. Neurosurg.* 62:115-19, 1985.
 119. Besson, A. and Saegesser, F.: *Color Atlas of Chest Trauma and Associated Injuries*. Oradell, NJ, Medical Ergonomics Books, 1983, vol. 1,2.
 120. Symbas, P.: Fundamentals of clinical cardiology-Cardiac trauma. *Am. Heart J.* 92:387-96, 1976.
 121. Viano, D.: Biomechanics of Impact Trauma-Chest: Anatomy, Types and Mechanisms of Injury, Tolerance Criteria and Limits, and Injury Factors. *Proceedings of the American Association for Automotive Medicine Conference*. Copper Mountain, CO, October 1984.
 122. Liedtke, A. and Demuth, W.: Non-penetrating cardiac injuries: A collective review. *Am. Heart J.* 86:687-97, 1983.
 123. Hess, R., Weber, K., and Melvin, J.: Review of Research on Thoracic Impact Tolerance and Injury Criteria Related to Occupant Protection. In: *Occupant Crash Interaction with the Steering System*, Viano, D.C., editor. *Society of Automotive Engineers. Int. Congress and Exposition*. Paper #820480. Detroit, February 1982, pp. 93-119.
 124. Lau, V.K. and Viano, D.C.: Influence of impact velocity and chest compression on experimental pulmonary injury severity in rabbits. *J. Trauma* 21:1-22-28, 1981.
 125. Zehnder, M.A.: Accident mechanism and accident mechanics of the aortic rupture in the closed thorax trauma. *Thoraxchir. Vasc. Chir.* 8:47-65, 1960.
 126. Lau, V.K. and Viano, D.C.: An experimental study on hepatic injury from belt-restraint loading. *Aviat. Space Environ. Med.* 52:611-17, 1981.
 127. Maltha, J. and Stalnaker, R.L.: Development of a Dummy Abdomen Capable of Injury Detection in Side Impacts. *Proceedings of the Twenty-Fifth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #811019. Davis, CA, September 1981, pp. 651-82.
 128. Melvin, J.W., Stalnaker, R.L., Roberts, V.L., et al.: Impact Injury Mechanisms in Abdominal Organs. *Proceedings of the Seventeenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #730968. Oklahoma City, OK, 1973, pp. 115-26.
 129. Neathery, R.F.: Analysis of Chest Impact Response Data and Scaled Performance Recommendations. *Proceedings of the Eighteenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #74188. Ann Arbor, MI, December 1974, pp. 459-94.
 130. Kroell, C.K., Pope, M.E., and Viano, D.C.: Interrelationship of Velocity and Chest Compression in Blunt Thoracic Impact. *Proceedings of the Twenty-*

- Fifth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #811016. Davis, CA, September 1981, pp. 549–82.
131. Rouhana, S.W., Lau, I.V., and Ridella, S.A.: Influence of velocity and forced compression on the severity of abdominal injury in blunt, nonpenetrating lateral impact. *J. Trauma* 25:490–500, 1984.
 132. Viano, D.C. and Artinian, C.G.: Myocardial conducting system dysfunctions from thoracic impact. *J. Trauma* 18:452–59, 1978.
 133. Kroell, C.K., Allen, S.D., Warner, C.Y., and Perl, T.R.: Interrelationship of Velocity and Chest Compression in Blunt Thoracic Impact to Swine II. *Proceedings of the Thirtieth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #861881. San Diego, CA, October 1986, pp. 99–121.
 134. Rouhana, S.W., Ridella, S.A., and Viano, D.C.: The Effects of Limiting Impact Force on Abdominal Injury: A Preliminary Study. *Proceedings of the Thirtieth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #861879. San Diego, CA, October 1986, pp. 65–79.
 135. Lasky, I.I., Siegel, A.W., and Nahum, A.M.: Automotive Cardio-Thoracic Injuries: A Medical-Engineering Analysis. *Automotive Engineering Congress, Society of Automotive Engineers*. Paper #680052. Detroit, MI, January 1968.
 136. Nahum, A.M., Gadd, C.W., Schneider, D.C., and Kroell, C.K.: Deflection of the Human Thorax Under Sternal Impact. *International Automobile Safety Conference, Society of Automotive Engineers*. Paper #700400. Detroit, MI, May 1970, pp. 797–807.
 137. Viano, D. and Levine, R., editors: *Symposium on Biomechanics and Medical Aspects of Lower Limb Injuries, Society of Automotive Engineers*. San Diego, October 1986.
 138. Evans, F. and Gaynor: A Review of Some Pioneering American Research Bioengineering. *Winter Annual Meeting of the American Society of Mechanical Engineers*. Washington, D.C., November 1981.
 139. Viano, D.C.: Considerations for a Femur Injury Criterion. *Proceedings of the Twenty-First Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #770925. New Orleans, October 1977, pp. 443–73.
 140. Viano, D.C. and Stalnaker, R.L.: Mechanisms of femoral fracture. *J. Biomech.* 13:701–16, 1980.
 141. Viano, D.C., Culver, C.C., Haut, R.C., et al.: Bolster Impacts of the Knee and Tibia of Human Cadavers and an Anthropomorphic Dummy. *Proceedings of the Twenty-Second Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #780896. Ann Arbor, MI, October 1978, pp. 401–28.
 142. Viano, D.C.: Femoral Impact Response and Fracture. *Proceedings of the 1980 International Conference on the Biokinetics of Impact*. Birmingham, England, 1980, pp. 261–72.
 143. Noyes, R. and Grood, E.S.: The strength of the anterior cruciate ligament in humans and rhesus monkeys. *J. Bone Joint Surg.* 58A:1074–82, 1976.
 144. Epstein, H.: Traumatic dislocations of the hip. *Clin. Orthop. Related Res.* 92:116–41, 1973.
 145. Viano, D.C. and Khalil, T.B.: Investigation of Impact Response and fracture of the Human Femur by Finite Element Modeling. *Nat. Automobile Engineering and Manufacturing Meeting, Society of Automotive Engineers*. Paper #760773. Dearborn, MI, October 1976, pp. 53–60.
 146. Yamada, H.: *Strength of Biological Materials*. Baltimore, Williams and Wilkins, 1970.
 147. King, J.J., Fan, R.S., and Vargovick, R.J.: Femur Load Injury Criteria. *Proceedings of the Seventeenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #730984. Oklahoma City, November 1973, pp. 509–24.

148. Hirsch, G. and Sullivan, L.: Experimental knee joint fractures-A preliminary report. *ACTA Orthop. Scand.* 36: 391-99, 1965.
149. Horsch, J.D. and Patrick, L.M.: Cadaver and Dummy Knee Impact Response. *Nat. Automobile Engineering and Manufacturing Meeting, Society of Automotive Engineers.* Paper #760799. Detroit, October 1976.
150. Frankel, V.H. and Nordin, M.: *Basic Biomechanics of the Skeletal System.* Philadelphia, Lea & Febiger, 1980.
151. Ghista, D., editor: *Osteoarthritis-mechanics.* New York, Hemisphere Pub., McGraw-Hill, 1982.
152. Fung, Y.C.: *Biomechanics-Mechanical Properties of Living Tissues.* New York, Heidelberg, Berlin, Springer-Verlag, 1981.
153. Fung, Y.C., Perrone, N., and Anliker, M., editors: *Biomechanics-Its Foundations and Objectives.* Englewood Cliffs, NJ, Prentice-Hall, 1972.
154. Ghista, D.N., editor: *Human Body Dynamics: Impact, Occupational, and Athletic Aspects.* Oxford, Clarendon Press, 1982.
155. Akkas, N., editor: *Progress in Biomechanics.* Alphen aan den Rijn, The Netherlands, Sijthoff & Noordhoff, 1979.
156. Kneighbaum, E. and Barthels, K.M.: *Biomechanics-A Qualitative Approach for Studying Human Movement.* Minneapolis, Burgess, 1981.
157. Reul, H., Ghista, D.N., and Rau, G.T. editors: *Perspectives in Biomechanics-Volume 1, Part A and B.* Chur, Switzerland, Harwood, 1978.
158. Halpern, J. and Davis, J.W.: Effects of ethanol in trauma. *J. Emerg. Nursing* 8:261-3, 1982.
159. Liedtke, A.J. and DeMuth, W.E.: *Effects of Alcohol on Cardiovascular Performance After Experimental Non-penetrating Chest Trauma.* Pennsylvania State University College of Medicine and the Milton S. Hershey Medical Center.
160. House, E., Waller, P., and Stewart, J.: Blood Alcohol Level and Injury in Traffic Crashes. *Proceedings of the 26th American Association for Automotive Medicine.* Ottawa, Ontario, Canada, October 1982, pp. 349-74.
161. Brodner, R.A., Van Gilder, J.C., and Collins, W.F.: Experimental spinal cord trauma: Potentiation by alcohol. *J. Trauma* 21:124-9, 1981.
162. Flamm, E.S., Demopoulos, H.B., Seligman, M.L., et al.: Ethanol potentiation of central nervous system trauma. *J. Neurosurg.* 46:328-35, 1977.
163. Mohan, D. and Melvin, J.: Failure properties of passive human aortic tissue I-Uniaxial tension tests. *J. Biomech.* 15:887-902, 1982.
164. Mohan, D. and Melvin, J.: Failure properties of passive human aortic tissue II-biaxial tension tests. *J. Biomech.* 16:31-44, 1982.
165. Waller, J.: Nonhighway injury fatalities-I. The roles of alcohol and problem drinking, drugs and medical impairment. *J. Chron. Dis.* 25:33-45, 1972.
166. *Human Tolerance to Impact Conditions as Related to Motor Vehicle Design.* Handbook Supplement J885. Warrendale, PA, Society of Automotive Engineers, 1980.
167. Hodgson, V.R. and Thomas, L.M.: Comparison of Head Acceleration Injury Indices in Cadaver Skull Fractures. *Proceedings of the 15th Stapp Car Crash Conference, Society of Automotive Engineers.* Paper #710854. Los Angeles, November 1971, pp. 190-206.
168. Blizard, J.R. and Howitt, J.S.: Development of a Safer Nonlacerating Automobile Windshield. *Midyear Meeting Society of Automotive Engineers.* Paper #690484. Chicago, May 1969.
169. Pickard, J., Brereton, P.A., and Hewson, A.: An Objective Method of Assessing Laceration Damage to Simulated Facial Tissues. *Proceedings of the Seventeenth Conference of American Association of Automotive Medicine.* Oklahoma City, November 1973, pp. 148-65.
170. Kay, S.E., Pickard, J., and Patrick, L.M.: Improved Laminated Windshield with Reduced Laceration Properties. *Proceedings of the Seventeenth*

- Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #730969. Oklahoma City, November 1973, pp. 127-70.
171. Mertz, H.J. and Patrick, L.M.: Strength and Response of the Human Neck. *Proceedings of the Fifteenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #710855. Los Angeles, November 1971, pp. 207-55.
 172. Mertz, H.J. and Patrick, L.M.: Investigation of the Kinematics and Kinetics of Whiplash. *Proceedings of the Eleventh Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #670919. Los Angeles, October 1967, pp. 267-317.
 173. Mertz, H.J., Hodgson, V.R., Thomas, L.M., et al.: An assessment of compressive neck loads under injury-producing conditions. *Phys. Sports Med.* 6:95-106, 1978.
 174. Nyquist, G.W., Begeman, P.C., King, A.I., and Mertz, H.J.: Correlation of field injuries and GM Hybrid III dummy response for lap-shoulder belt restraint. *J. Biomech. Eng.* 102: 1982.
 175. Stapp, J.P.: Voluntary Human Tolerance Levels. In: *Impact Injury and Crash Protection*. Springfield, Thomas, 1970.
 176. Mertz, H.J. and Gadd, C.W.: Thoracic Tolerance to Whole-Body Deceleration. *Proceedings of the Fifteenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #710852. Los Angeles, November 1971, pp. 135-57.
 177. Viano, D.C., Schreck, R.M., and States, J.D.: Dive Impact Tests and Medical Aspects of a Seventy-Year-Old Stune Diver. *Proceedings of the Nineteenth Conference of the American Association for Automotive Medicine*. San Diego, November 1975, pp. 101-15.
 178. Melvin, J.W. Hohan, D., and Stalnaker, R.L.: Occupant Injury Assessment Criteria. *Society of Automotive Engineers. Automotive Engineering Congress*. Paper #750914. Detroit, October 1975.
 179. Patrick, L.M., Mertz, H.J., and Droell, C.K.: Cadaver Knee, Chest and head Impact Loads. *Proceedings of the Ninth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #670913. Los Angeles, October 1967, pp. 168-82.
 180. Powell, W.R., Odala, S.J., Advani, S.H., and Martin, R.B.: Cadaver Femur Responses to Longitudinal Impacts. *Proceedings of Nineteenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #751168. San Diego, November 1975, pp. 561-80.
 181. Melvin, J.W., Stalnaker, R.L., Alem, N.M., Benson, J.B. and Mohan, D.: Impact Response and Tolerance of the Lower Extremities. *Proceedings of the Nineteenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #751159, San Diego, November, 1975, pp. 543-60.
 182. *Anthropomorphic Test Dummy*, Volumes I, II, III. Final Report No. DOT-HS-299-3-569. Prepared by General Motors Corporation for the National Highway Traffic Safety Administration. December, 1973.
 183. Tennant, J.A., Jensen, R.J., and Potter, R.A.: GM-ATD 502 Anthropomorphic Test Dummy-Development and Evaluation. *Proceedings of the Fifth International Technical Conference on Experimental Safety Vehicles*. London, June 1974, pp. 446-62.
 184. Hubbard, R.P.: Anthropomorphic Basis of the GM-ATD 502 Crash Test Dummy. *Society of Automotive Engineers Automotive Engineering Congress*. Paper #750429. Detroit, February 1975.
 185. Hubbard, R.P. and McLeod, D.G.: A Basis for Crash Dummy Skull and Head Geometry. In: *Human Impact Response-Measurement and Simulation*. New York, Plenum, 1973.
 186. Hubbard, R.P. and McLeod, D.G.: Definition and Development of a Crash Dummy Head. *Proceedings of Eighteenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #741193. Ann Arbor, MI, December 1974, pp. 599-628.
 187. Horsch, J.D. and Culver, C.C.: A

- Study of Driver Interactions with an Inflating Air Cushion. *Proceedings of Twenty-Third Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #791029. San Diego, October 1979, pp. 797-824.
188. Gadd, C.W. and Patrick, L.: System Versus Laboratory Impact Tests for Estimating Injury Hazard. *Society of Automotive Engineers Congress*. Paper #680053. Detroit, January 1968.
189. Marquis, D.P.: Second Generation Energy Absorbing Column with Locking Features. *Society of Automotive Engineers Automotive Engineering Congress*. Paper #70002. Detroit, January 1970.
190. Skeels, P.C. and Hanson, H.L.: The General Motors Energy Absorbing Steering Column: A Case History. *Proceedings of the Tenth Stapp Car Crash Conference, Society of Automotive Engineers*. Paper #660785. Holloman Air Force Base, NM, November 1966, pp. 1-13.
191. Stapp, J.P.: Past, Present, and Future of Biomechanics at Wayne State University. *Proceedings of the Eighth Stapp Car Crash Conference, Society of Automotive Engineers*. Detroit, October 1964, pp. 391-99.
192. Kroell, C.K. and Patrick, L.M.: A New Crash Simulator and Biomechanics Research Program. *Proceedings of the Eighth Stapp Car Crash Conference, Society of Automotive Engineers*. Detroit, October 1964, pp. 185-228.
193. Hobbs, C.A. and Mills, P.J.: Injury Probability for Car Occupants in Frontal and Side Impacts. In: *Transport and Road Research Laboratory Report 1124*. Growthorne, England, Transport and Road Research Laboratory, 1984.
194. Daniel, R.P.: A Bio-Engineering Approach to Crash Padding. *Society of Automotive Engineers Automotive Engineering Congress*. Paper #680001. Detroit, January 1968.
195. Lundstrom, L.C. and Cichowski, W.G.: Field Experience with the Energy Absorbing Steering Column. *International Automotive Engineering Congress*. Paper #690183. Detroit, January 1969.
196. Patrick, L.M. and Van Kirk, D.J.: Correlation of Accident and Laboratory Impacts to Energy-Absorbing Steering Assemblies. *International Automotive Engineering Congress*. Paper #690185. Detroit, January 1969.
197. Mathematical Simulation of Occupant and Vehicle Kinematics. *Society of Automotive Engineers. Government/Industry Meeting and Exposition*. Publication, P-146. Washington, D.C., May 1984.